Final report

OWMC
Ontario
Waste Management
Corporation

A provincial crown corporation

ENVIRON
Counsel in Health and Environmental Science

SITE ASSESSMENT
PHASE 4B: RISK ASSESSMENT
VOLUME 1

Prepared for
The Ontario Waste Management Corporation

Submitted by

ENVIRON Corporation

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January 29, 1988

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Re: Site Specific Risk Assessment, Volumes 1 and 2

Dear Sirs:

We are pleased to submit our Site Specific Risk Assessment, Volumes 1 and 2. Volume 1 consists of the technical report; Volume 2 contains appendices of supporting technical information.

Sincerely,

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EXECUTIVE SUMMARY

PURPOSE OF THE REPORT

The most highly ranked goal in establishing the proposed OWMC Hazardous Waste Management Facility is to minimize risks to human health. The second most highly ranked goal is to minimize risks to the environment. The principles of risk assessment were therefore applied by OWMC to develop a quantitative estimate of the potential human health and environmental risks associated with facility operations. The results of the risk assessment provide a basis for:

- Judging the acceptability of potential risks associated with the operation of the proposed OWMC Facility;
- Evaluating the suitability of the preferred site for the facility;
- Identifying elements of the facility design and operation which could be modified, if necessary, to reduce risks to human health and the environment; and
- Developing management strategies, such as monitoring programs, to ensure the safe operation of the proposed facility.

In Phase 4A of the OWMC Facilities Development Process, ENVIRON conducted a generic risk assessment to assist in the selection of a preferred site for the proposed facility. The term "generic" was used to indicate that the Phase 4A Risk Assessment was based on a single set of generalized parameters applicable to all candidate sites. The results of that analysis were considered, along with inputs from a variety of other studies conducted by OWMC, in selecting a site in West Lincoln Township. In Phase 4B, it was possible to refine the risk analysis to better reflect the specific characteristics of the area surrounding the West Lincoln site, and to incorporate additional information with respect to the proposed design and operation of the facility. The assessment was also expanded to include additional exposure pathways and refined to provide a more complete evaluation of the potential risks associated with facility operations.

SCOPE AND METHOD

The Phase 4B Risk Assessment addressed the potential for adverse effects to both human health and the environment. Consideration was given to long-term exposures associated with normal facility operations and short-term exposures associated with both on-site upsets and accidents, and off-site transportation accidents.

The Phase 4B Risk Assessment was expanded to include an evaluation of routine emissions from the OWMC Facility that had not been considered in the Phase 4A generic analysis. In Phase 4A, incinerator stack and fugitive emissions were examined in developing estimates of potential long-term human health risks. The Phase 4B assessment includes an evaluation of potential contaminant releases from the engineered landfill and the evaporation unit of the physical/chemical treatment plant as well. These emission sources could not be considered in Phase 4A since sufficiently detailed information on facility design and operation was not available at the time the generic risk assessment was performed.

The Phase 4B Risk Assessment also addresses specific public concerns raised since the preparation of the Phase 4A report. For example, the human health risks associated with the inhalation of air, and the ingestion of beef, milk, pond water, cistern water, and leafy vegetables were evaluated in Phase 4A. The scope of the Phase 4B analysis has been expanded to include an examination of additional human exposure pathways, such as the consumption of various fruits and vegetables, pork, poultry, and eggs, incidental ingestion of soil, and dermal absorption of contaminants in soil.

For each pathway examined, exposures were assessed using a four step process: (1) identification of significant sources of contaminant release to the environment; (2) characterization of the nature, magnitude, frequency, and duration of each selected release; (3) modeling of contaminant fate and transport in the environment; and (4) estimation of the total systemic dose received by the exposed human or environmental receptor. The risk associated with both long-term (chronic) and short-term (acute) exposures were evaluated.

In the human health risk assessment, estimates of the additional lifetime cancer risk associated with chronic exposures to carcinogens were developed. The cancer risks associated with exposures to individual carcinogens were added to calculate the total cancer risk for each human exposure pathway examined. For chronic exposures to non-carcinogens, the predicted human dose was compared to a toxicologically safe level referred to as the Acceptable Daily Intake (ADI). The ratio of the ADI to the predicted level of exposure is defined in this report as the Adjusted Margin of Safety (AMOS). The AMOS values were aggregated for each organ of the body to provide an estimate of the daily burden placed on an organ due to exposure to a mixture of chemicals.

Acute exposures were evaluated in a manner similar to that for chronic exposures to non-carcinogens. In evaluating an acute exposure, a toxicologically safe level derived from a short-term study was divided by the predicted human dose to calculate an acute AMOS value. The AMOS values were used to predict whether or not any effect would be experienced in the exposed human population. In cases where some type of effect would be expected, the type of effect was characterized.

In the environmental risk assessment, safe levels for the acute and chronic exposure of animal and plant species to the chemicals of concern were derived from the scientific literature. These safe exposure levels were then divided by the predicted systemic dose to develop AMOS values. The AMOS values were used to determine whether adverse effects to the environment are likely to be caused by the operation of the proposed OWMC Facility.

PREMISES AND POSTULATES

In many cases, information used in performing the risk assessment was supplied to ENVIRON by OWMC and its consultants. For example, an estimate of the characteristics of the wastes to be received by the facility was developed by OWMC; information on the potential magnitude of emissions from the facility was provided by engineering consultants to OWMC.

The risk assessment was undertaken for a proposed facility capacity of 150,000 tonnes per annum (t/a) of special waste, with a sensitivity analysis performed to evaluate a possible expansion to 300,000 t/a. A 70-year period of operation was assumed. Risk estimates were based on preliminary plans for the design and operation of the facility and did not account for the mitigation of potentially adverse effects. Such mitigation measures are being considered by OWMC in developing the final design for the facility.

In an effort to ensure that potential risks associated with the operation of the OWMC were not underestimated, an approach based on the use of conservative assumptions was adopted in the risk assessment. These assumptions are termed conservative in that they tend to lead to an overstatement of the risks associated with emissions from the proposed OWMC Facility. The results of the assessment should therefore be considered to represent a conservative estimate of risk.

STATUS OF DATA

Data used in the risk assessment were provided from two principal sources: (1) published literature on the behavior of chemicals in the environment, specifically their transport, fate and toxicity; and (2) consultant studies undertaken for

OWMC. These data were used to develop estimates of the nature and magnitude of emissions from the proposed facility, and an understanding of the potential effects these releases may have on human health and the environment.

Information on types and quantities of chemicals to be managed by the proposed facility were based primarily on a survey of 1,000 industries in Ontario. In some cases, only a limited amount of quantitative data on specific constituents of the wastes was available from this source. In addition, some contaminants are identified only in a descriptive or generic manner; for example, the largest organic contaminant projected to be received at the facility is identified only as "oil and grease". A supplementary data base was therefore used to characterize the potentially hazardous components of oil and grease. In order to further account for a lack of complete waste characterization data, a procedure was developed to represent the potentially hazardous portion of the wastes to be received at the facility by specific, well-defined chemicals. In this way, the risks associated with the total quantity of potentially hazardous waste were evaluated, and the effects of changes in waste composition over time were taken into account to the extent possible.

The risk assessment relied on both human epidemiology studies and animal experiments to evaluate the potential adverse impacts that may be associated with exposures to chemicals emitted from the OWMC Facility. While limitations and uncertainties are associated with the use of such data, the application of safety factors and conservative mathematical models provides a systematic methodology for scientifically evaluating the toxicity of the chemicals of concern.

Overall, the use of conservative assumptions and safety factors in the Phase 4B assessment has allowed for the development of risk estimates that are not likely to underestimate and may well significantly overestimate potential human health and environmental risks associated with the operation of the proposed facility.

MAIN FINDINGS

HUMAN HEALTH RISK ASSESSMENT

The routine atmospheric release of non-carcinogenic chemicals from the proposed OWMC Facility is not predicted to pose a significant risk to the off-site community. For the non-carcinogens, the estimated levels of chronic exposure would not be anticipated to produce any type of adverse human health effects.

For carcinogens, the risk of an individual developing cancer as a result of exposures to routine facility emissions is predicted to be much lower than the risk posed by many normally and routinely experienced exposures (e.g., medical diagnostic x-rays, background radiation). Furthermore, the cancer risk associated with facility operations would be many times lower than the current level of cancer risk in Ontario. The cancer risks posed by the proposed facility fall well within the range that has been judged acceptable by regulatory bodies and decision-makers.

The impact that the OWMC engineered landfill may have on ground water resources has also been examined. This landfill will receive only solidified treatment residues and solid inorganic waste. It has been estimated that the extensive clay layer beneath the preferred site will isolate chemicals in the leachate from the ground water for a period of at least hundreds or thousands of years. Some chemicals, in fact, would likely be isolated from the aquifer for periods approaching millions of years. Furthermore, the solidification process to be used at the OWMC facility will effectively stabilize many contaminants, especially metals, thereby limiting concentrations in the leachate. Based on these factors, it is unlikely that the migration of metals from the landfill will pose a significant risk to public health. In addition, although it is not currently possible to accurately predict the environmental fate of non-metals present in the leachate, the analysis conducted indicates that various attenuation mechanisms would likely prevent significant ground water contamination.

A detailed hazard analysis of the proposed facility is currently being conducted by OWMC. The hazards associated with those events identified as posing a potentially significant risk to public health will be examined by the completion of that study. To date, however, the consequences of a number of on-site accidents and upsets have been evaluated. Of these events, only the tank farm fire appears to have the potential to cause significant adverse health effects in the exposed off-site population; the levels of exposure, however, are not likely to cause death or incapacitating illness. This event has been estimated to occur with a frequency not greater than once in one hundred years if the storage tanks are not segregated; if the tanks are segregated, the frequency would be reduced to approximately once in every 700 to 1 300 years. The consequence and/or frequency of a tank farm fire could be further reduced if other mitigation measures, e.g. tank insulation, are incorporated into the facility design.

Potential public health risks associated with off-site transportation accidents were also evaluated. The assessment of a transportation spill onto the roadway indicates that potentially significant human health effects could be experienced at the point of maximum exposure. Similarly, stream water contaminated by a spill of special waste could pose a significant risk to human health under certain conditions.[1] Potentially hazardous transportation spills are, however, expected to occur relatively infrequently. For example, a potentially hazardous spill onto the roadway is predicted to occur only once in over 130 years. A spill of any type into Twenty Mile Creek (the largest stream along the local access route) would be expected to occur less than once in 400 years.

ENVIRONMENTAL RISK ASSESSMENT

Routine atmospheric releases of chemicals from the proposed OWMC Facility are not expected to pose a significant risk to environmental receptors in the vicinity of the proposed facility. Of the on-site upsets and accidents evaluated, only the tank farm fire appears to have the potential to cause significant adverse effects on livestock and wildlife located near the proposed site. As previously noted, such an event would be expected to occur with a frequency of less than once in 100 years if the tanks are unsegregated, and approximately once in 700 to 1 300 years if the tanks are segregated.

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[1] It should be noted that the risks associated with off-site spills of waste to be received at OWMC are similar in many respects to the risks associated with the transport of many common materials (e.g., gasoline and chemical products).

The assessment of a transportation spill onto a roadway indicates that adverse impacts on livestock and wildlife could occur at the point of maximum exposure. It is also likely that such a spill would adversely effect crops and natural vegetation in the area of the spill. Surface waters contaminated by a spill of special waste would be likely to pose a significant risk to aquatic life. However, as previously noted, transportation accidents leading to potentially hazardous spills are expected to occur infrequently. In addition, mitigation measures proposed by OWMC, such as quick emergency response, could reduce both the frequency and consequence of hazardous spill events.

CONCLUSIONS AND RECOMMENDATIONS

The site-specific risk assessment conducted in Phase 4B indicates that routine emissions to the atmosphere are not likely to cause adverse effects in exposed human or environmental receptors. Although the potential for some type of adverse effects may be associated with a tank farm fire or a spill resulting from a transportation accident, such events are expected to occur infrequently, and mitigation measures can be employed to further reduce potential risks.

Although the Phase 4B assessment indicates that the risks associated with routine operation of the proposed OWMC Facility are likely to be small, it is recommended that OWMC undertake monitoring programs to confirm the results of the analysis and assist in identifying any contingency measures that may be required. Such programs should include periodic sampling and analyses for specific contaminants emitted from the incinerator and evaporator stacks. It is further recommended that sampling and analysis for volatile organic chemicals be performed on-site to ensure that potentially hazardous levels of fugitive emissions are not generated. As the data from the analysis of incinerator, evaporator, and fugitive emissions become available, the risk assessment can be revised to reflect actual operations. It should be noted that OWMC has already begun the development of on-site monitoring programs such as those recommended in this report.

It is also recommended that a biomonitoring program be conducted to address public concerns regarding the operation of the proposed facility. Such a program is currently being developed by OWMC, and will encompass the study of vegetation, crops, soils, and livestock. Observations made during the pre-operational stage will serve as a baseline to which observations made during the operational phase of the facility can be compared.

Data for predicting the human health risks that may be associated with landfill operations are sparse. Efforts have been made in the assessment to provide a conservative estimate of the quantities of potentially hazardous chemicals that may be present in leachate. It is recommended, however, that additional studies be undertaken to better characterize leachate quality and the fate and transport of chemicals released from the landfill.

It is appropriate to develop monitoring and contingency measures for any landfill, including the one proposed by OWMC, to ensure the protection of human health. Therefore, a monitoring program designed to provide the capability for early detection of the potential for significant ground water contamination should be adopted and implemented by OWMC. Based on the results of such monitoring, contingency measures, such as leachate collection, or the institution of purge wells to collect ground water for treatment, could be implemented if necessary. It is also recommended that a specific protocol be adopted to identify and exclude from the landfill any waste which threatens to significantly degrade ground water quality.

As previously noted, a detailed hazard analysis for the facility is currently being conducted by OWMC. This study has been undertaken to identify upsets and accidents which may pose a significant risk to public health. It is recommended that OWMC consider modifications to the design and operation of the facility to limit the hazards associated with those events which may pose a significant risk to either on-site personnel or the off-site community. It is further recommended that detailed emergency response procedures be prepared by OWMC following an evaluation of the results of the hazard analysis. These procedures should address both on-site and off-site responses, such as community notification in the event of a potentially serious accident and, if necessary, evacuation. Similar emergency response procedures should be developed to address potential off-site transportation accidents.

CHAPTER 1

INTRODUCTION

1.1 BACKGROUND AND PURPOSE

The Ontario Waste Management Corporation (OWMC) is currently engaged in a Facilities Development Process to establish a special waste management facility for the Province of Ontario. As shown in Figure 1, this process consists of five phases, culminating in public hearings required under the Environmental Assessment Act. These hearings will take place prior to construction of the proposed facility.

A major objective of the Facilities Development Process was the selection of a preferred site for the proposed OWMC Facility. The selection program was begun in Phase 1, where information on waste types and available treatment technologies was collected. In Phase 2, candidate regions were chosen; in Phase 3, eight potentially suitable sites were identified. In Phase 4A, these eight sites were compared on the basis of three sets of factor groupings: Risks, Impacts, and Costs (M.M. Dillon 1985).

Figure 1
The OWMC Facilities Development Process

PHASE I

INFORMATION GATHERING

PHASE II

CHOOSE CANDIDATE REGION
&
GENERIC TECHNOLOGIES

PHASE III

A. CHOOSE CANDIDATE AREAS

B. CHOOSE CANDIDATE SITES

PHASE IV

A. CHOOSE:
PREFERRED SITE(S)
TECHNOLOGIES
CONCEPTUAL DESIGNS

B. SITE SPECIFIC STUDIES & DESIGNS

PHASE V

PUBLIC HEARINGS
UNDER
ENVIRONMENTAL ASSESSMENT ACT

As part of the Phase 4A activities, an assessment was performed to provide the data required to compare potential human health risks associated with the various siting options (ENVIRON, 1985). This risk assessment was termed "generic" since the analysis was based on a single set of general parameters applicable to all of the candidate sites. The results of this risk assessment were considered, along with inputs from a variety of other studies conducted by OWMC, in selecting a preferred site in West Lincoln Township.

In conjunction with the Phase 4B site-specific studies and designs for the OWMC Facility, ENVIRON has refined the Phase 4A generic risk assessment to reflect more closely the specific characteristics of the West Lincoln site. The Phase 4B risk assessment has also been expanded to include a consideration of information on the design and operation of the proposed facility that was not available at the time the generic analysis was conducted. The purpose of the Phase 4B site-specific risk assessment is to provide OWMC with clearer understanding of the potential risks associated with the operation of the proposed special waste management facility at the West Lincoln site. The assessment will thereby provide a basis for judging the acceptability of the potential risks, and for developing various management strategies for reducing risks as necessary.

1.2 SCOPE OF THE RISK ASSESSMENT

The Phase 4B risk assessment has been conducted to provide quantitative estimates of the risks associated with exposures to chemicals that may be released to the environment during the operation of the proposed OWMC Facility. Both risks to the potentially exposed human population and to livestock, wildlife, and other environmental receptors in the site vicinity have been examined.

A variety of contaminant releases which may occur during normal operation of the proposed OWMC Facility have been considered in the risk assessment. Those routine emissions which are believed to pose the greatest risk to public health have been quantitatively evaluated. These releases are listed in Table 1-1.

As shown in Table 1-1, the Phase 4B Risk Assessment includes a consideration of potential contaminant releases from both the engineered landfill and the evaporation unit at the physical/chemical treatment plant. It was not possible to consider either of these releases in the earlier Phase 4A generic risk assessment due to limitations in available data with respect to site characteristics and the design and operation of the proposed facility. It should be noted that

TABLE 1-1

ROUTINE EMISSION SOURCES EVALUATED IN THE RISK ASSESSMENT

- Incinerator stack emissions
- Evaporator stack emissions
- Fugitive releases associated with
  - Bulk unloading and transfer,
  - Routine leaks and spills,
  - Drum handling,
  - Waste storage, and
  - Physical/Chemical treatment processes.
- Leachate from the engineered landfill.

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the evaluation of the risks associated with releases of leachate from the engineered landfill is not complete due to a lack of information on the fate and transport of chemicals in the 15-20 meter clay layer which lies between the landfill and the aquifer beneath the site. Studies to better characterize the behavior of chemicals in this clay layer over time are currently being performed.

Exposures resulting from various accidents and upsets have also been examined in the risk assessment. Most of these events were previously evaluated in Phase 4A (ENVIRON, 1985), although the assessment has been expanded in Phase 4B to consider the risks associated with a transportation accident resulting in a spill of special wastes into a stream. The sources of short-term contaminant releases examined in the risk assessment are shown in Table 1-2.

For each emission source evaluated, those exposure pathways believed to be most significant were examined to develop quantitative estimates of potential risks. For long-term contaminant emissions to the atmosphere, a variety of human exposure pathways, including inhalation of air, ingestion of food, water, and soil, and dermal contact with soil were evaluated. A variety of exposure pathways, including inhalation and ingestion of food and water, were similarly analyzed in the environmental risk assessment. For routine

TABLE 1-2

SHORT-TERM EMISSION SOURCES EVALUATED IN THE RISK ASSESSMENT

- A fire in the halogenated organic waste tank farm resulting in the boiling of a storage tank containing solvent wastes. This could lead to the short-term release of halogenated organics to the atmosphere.

- The mixing of incompatible wastes, resulting in the generation of a toxic gas, hydrogen sulfide (H_2S).

- The failure of the scrubbing system designed to reduce acid gas emissions from the incinerator stack.

- pH excursions during the treatment of cyanide-containing wastes, resulting in the generation of a toxic gas, hydrogen cyanide (HCN).

- pH excursions during sulfide precipitation of heavy metals, resulting in the generation of a toxic gas, hydrogen sulfide (H_2S).

- Transportation spill leading to the release of special wastes
  - -- onto the roadway, resulting in air contamination; or
  - -- into a stream, resulting in water contamination.

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releases to ground water, the human health risks associated with the ingestion of drinking water were examined. Ingestion of ground water was not examined in the environmental risk assessment since it was judged to represent a relatively insignificant pathway of exposure for livestock, crops, and wildlife.

The evaluation of releases that may occur as the result of upsets, malfunctions and accidents was restricted to a consideration of short-term exposures in both the human health and environmental risk assessments. OWMC is committed to performing remedial measures as necessary to prevent long-term exposures to unacceptable levels of chemicals released during such events. Thus, the most significant short-term exposures were considered to be those that might occur before any remedial activities could be implemented.

1.3 RELATIONSHIP TO OTHER PHASE 4B ACTIVITIES

Many inputs to the risk assessment have been produced by other studies commissioned by OWMC. For example, potential sources of contaminant release to the environment were identified through a review of the design of the proposed facility (Monenco 1987a); the characterization of the special wastes to be received at the facility was based primarily on information compiled by CANVIRO Consultants Ltd. (1985); and the release rates for contaminants from certain emission sources were derived using information provided by the Ontario Research Foundation (1987) and Monenco (1987b). The various inputs to the Phase 4B risk assessment are summarized in Table 1-3.

The results of the risk assessment provide a basis with which to evaluate the suitability of the preferred West Lincoln site. Furthermore, the risk assessment will assist in identifying any elements of the facility design and operation which could be, if necessary, modified to reduce risks to human health and the environment. The analysis will also be used by OWMC in the development of monitoring programs and risk management strategies to help ensure that the operation of the proposed facility will not pose unacceptable human health or environmental risks.

1.4 ORGANIZATION OF THE REPORT

The risk assessment report is organized into two volumes. The first volume consists of twelve chapters that outline the general approaches used in the risk assessment and present results and conclusions. The second volume contains fifteen

TABLE 1-3

INPUTS TO RISK ASSESSMENT FROM OTHER CONSULTANT STUDIES

Input Data	Source	Analysis undertaken by ENVIRON
Waste profile	OWMC	Estimation of the quantities of various types of waste to be received at the facility
Facility design	Monenco	Identification of potential release sources
Facility operation	Monenco	Estimation of contaminant releases from evaporation unit
Waste stream characteristics	CANVIRO	Selection of surrogate chemicals
On-site atmospheric emissions and calculations	Monenco	Estimation of chemical-specific emissions
Empirical emissions data	ORF EEC/Von Roll	Estimation of chemical-specific emissions
Definition of facility upset and accident conditions	M.M. Dillon Monenco	Estimation of chemical-specific emissions
Tank farm fire frequency	Battelle	Estimation of the probability of a tank farm fire
Transportation spill frequency	M.M. Dillon	Estimation of the probability for spill events
Air dispersion modeling	ORF	Estimation of chemical concentrations in air
Chemical deposition rates	ORF	Estimation of chemical concentrations in rates cisterns and ponds, surface water, plants, and soil
Soil characteristics	Ecologistics	Estimation of chemical concentrations in soil

continued . . .

TABLE 1-3 (continued)

INPUTS TO RISK ASSESSMENT FROM OTHER CONSULTANT STUDIES

Input Data	Source	Analysis undertaken by ENVIRON
Livestock and crop production data	Ecologistics	Estimation of chemical concentrations in vegetables, meat and milk
Partitioning of chemicals in the landfill	Wastewater Technology Centre	Estimation of chemical concentrations in landfill leachate

technical appendices that provide details on the methodologies used in conducting the assessment.

The contents of each chapter in Volume 1 are summarized below.

- Chapter 1, Introduction, provides an overall description of the scope and objectives of the risk assessment.
- Chapter 2, The Risk Assessment Process, provides a description of the principles of risk assessment, and the application of these principles in evaluating the proposed OWMC special waste management facility. The limitations and uncertainties associated with the risk assessment process are also discussed.
- Chapter 3, The Existing Environment, describes the environment in the immediate vicinity of the proposed West Lincoln site. Background levels of contamination in soil, air, and water are also provided.
- Chapter 4, Characterization of Wastes, identifies the sources of data used to define the nature and quantity of the wastes to be received at the proposed OWMC Facility. The general approaches used in characterizing the wastes for the purposes of the risk assessment are also discussed.
- Chapter 5, Routine Emissions to the Atmosphere, discusses atmospheric emissions which are expected to occur during normal operation of the proposed facility. These include incinerator stack, evaporator, and fugitive emissions. The nature and magnitude of these emissions, and the potential pathways for human exposure to the chemicals released, are described.
- Chapter 6, Releases from the Engineered Landfill, outlines the approaches used in evaluating the potential release of chemicals in leachate generated in the OWMC landfill.
- Chapter 7, Releases Associated with On-Site Accidents and Upsets, describes certain events representing deviations from normal operation of the OWMC

Facility. The upsets and accidents considered, and the potential for human exposure to the emissions, are discussed.

- Chapter 8, Releases Associated with Off-Site Transportation Accidents, contains a description of the transportation accident scenarios evaluated in the risk assessment for the proposed OWMC Facility. The nature and magnitude of the releases, and the potential for human exposure to the chemicals spilled, are discussed.

- Chapter 9, Assessment of the Toxicity of the Contaminants of Concern, discusses the approaches used in the risk assessment to evaluate the acute and chronic toxicity of the contaminants that may be released to the environment during facility operations.

- Chapter 10, Public Health Risks Associated with the Operation of the OWMC Facility, provides a summary of the human health risks calculated for each exposure pathway and for each source of release evaluated in the risk assessment.

- Chapter 11, The Environmental Risk Assessment, describes the general methodology used to evaluate potential impacts to livestock, wildlife, and other environmental receptors located in the vicinity of the proposed site. The results of this analysis are also discussed.

- Chapter 12, Conclusions and Recommendations, presents the results of the human health and environmental risk assessments, and discusses their significance in evaluating the proposed facility. Recommendations for the development of monitoring programs and risk management strategies are also provided.

A glossary of the technical terms used in the report is provided following Chapter 12.

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CHAPTER 2

THE RISK ASSESSMENT PROCESS

2.1 THE PRINCIPLES OF RISK ASSESSMENT

2.1.1 Introduction

Risk can be defined as the probability of injury, disease or death, and may be expressed in numerical terms over the range between zero (absolute certainty that harm will not occur) and one (absolute certainty that harm will occur). The level of risk associated with a chemical exposure is a function of the inherent toxicity of the chemical(s) involved and the route, duration, and magnitude of the dose received.

For some types of chemical exposures, such as accidental poisoning by household products, a significant body of historical data exists on the relationship between an exposure and the incidence of adverse health effects. Risks associated with exposures that do not produce immediately observable effects are much more difficult to evaluate. In such cases, the process of risk assessment can be used to provide a quantitative estimate of the increased probability of adverse human health effects.

As shown in Figure 2, a complete risk assessment consists of the following components:

- Exposure Assessment,
- Hazard Evaluation, and
- Risk Characterization.

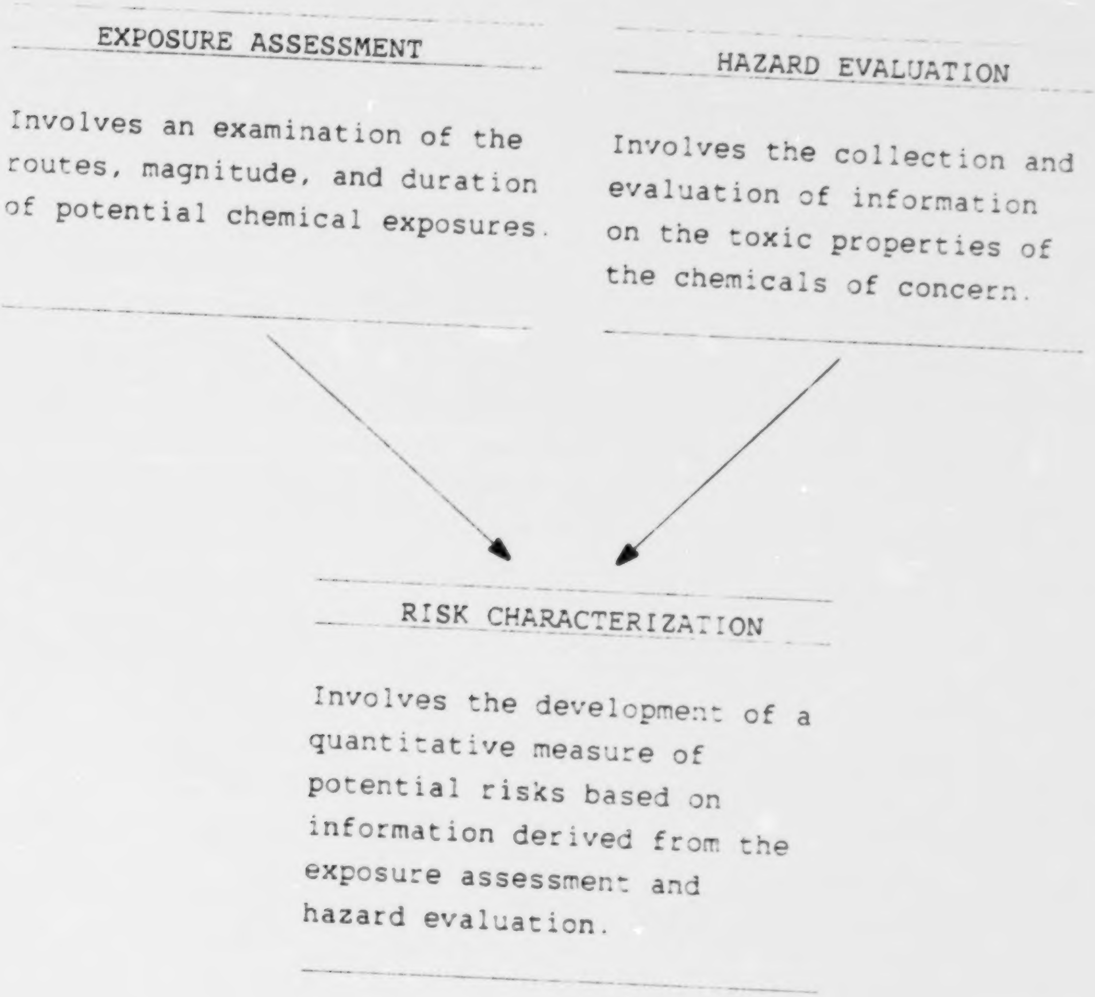
These three components of the risk assessment process are described briefly in the following sections.

2.1.2 EXPOSURE ASSESSMENT

The first component of risk assessment involves an examination of the routes, magnitude and duration of potential exposure to the chemical(s) of concern. Human exposure is typically assessed in terms of the dose of a particular substance taken into the body. For long-term exposures, the dose received is usually measured in milligrams of the chemical per kilogram of body weight per day. For exposures of shorter duration, the dose is more commonly measured in milligram of the chemical per kilogram of body weight. Short-term doses may also be assessed in terms of the contaminant concentration to which an individual is exposed, typically in units of milligram per

FIGURE 2

THE PRINCIPLES OF RISK ASSESSMENT



EXPOSURE ASSESSMENT

Involves an examination of the routes, magnitude, and duration of potential chemical exposures.

HAZARD EVALUATION

Involves the collection and evaluation of information on the toxic properties of the chemicals of concern.

RISK CHARACTERIZATION

Involves the development of a quantitative measure of potential risks based on information derived from the exposure assessment and hazard evaluation.

cubic meter or parts per million, and the duration of the exposure.

An evaluation of the three potential routes of human exposure (inhalation, ingestion, and dermal absorption) is often required to estimate the total human dose for the chemical(s) of concern. The significance of each of these three routes is dependent, in part, upon the magnitude of the release into the various environmental media, e.g., air, water and soil. Once the sources of release have been characterized, environmental fate and transport models can be used to estimate the concentration of the contaminant(s) in these environmental media. Certain standard conventions are then commonly applied to calculate the magnitude of human exposure (i.e., dose of each contaminant); these conventions assume average values for the intake of air, food, water, etc., and for the absorption of contaminants within the body.

2.1.3 HAZARD EVALUATION

Hazard is the inherent potential of chemical, physical or biological agents to cause some form of injury, disease or death under specified conditions of exposure. The hazard evaluation component of the risk assessment process involves the collection and evaluation of data on the inherent toxicity of the chemical(s) of concern. This information is obtained primarily from two sources: (1) epidemiological or clinical investigations, and (2) experimental studies involving laboratory animals or other biological systems.

Hazard evaluation provides information on the nature of the toxic effects associated with chemical exposures, and may also indicate which human sub-populations are particularly vulnerable to the adverse effects identified. The hazard evaluation also attempts to identify the relationship that exists between dose level and a given biological response. For example, the frequency with which toxic effects appear in an exposed population (i.e., risk) typically increases with increasing exposure or dose. The nature and severity of toxic effects may also change as exposure increases.

For the purposes of risk assessment, chemicals are usually divided into two categories: carcinogens (chemicals which may cause cancer) and non-carcinogens. Manifestation of most non-carcinogenic effects requires that a threshold level of exposure be exceeded. The starting point for risk assessment of chemicals that are assumed to operate by a threshold mechanism is the identification of the dose at which no effects are observed in a population of humans or experimental animals. This dose is frequently known as the "no-observed-effect-level" (NOEL). If data identifying a NOEL are unavailable, a "lowest-observed-effect-level" (LOEL) is sometimes used as the basis for determining the safe threshold dose.

In assessing the chronic effects of non-carcinogens, the experimentally-determined NOEL is typically divided by a "safety factor" to estimate an Acceptable Daily Intake (ADI). The application of safety factors is based on an understanding that the range of susceptibilities in a human population is much wider than in a genetically homogeneous test animal population; the safety factor is also used to offset the uncertainties associated with extrapolation of data from animals to humans.

The magnitude of the safety factor used to estimate an ADI is determined by the nature and quality of the experimental data; for example, in a well-conducted, chronic animal toxicity study in which the NOEL is carefully identified, the usual safety factor applied to calculate an ADI is 100. Where experimental data are lacking or limited, or when the experimental biological response is especially serious, a larger safety factor may be appropriate.

It is common practice in assessing the risk of carcinogens, however, to assume that some level of risk is associated with all finite exposures, i.e. no threshold exists. Therefore, it is usually necessary to predict low-dose risk for human exposure to carcinogens from high-dose/high-risk data derived from animal studies. This can be accomplished through the application of certain mathematical models to the dose-response data to produce an estimate of the excess cancer risk per unit dose. This value is commonly known as the Unit Cancer Risk (UCR). It should be noted, however, that some scientists and regulatory bodies believe that certain carcinogens require that a threshold level of exposure be exceeded to provoke a carcinogenic response (OMOE 1985).

For acute effects, an assessment of health risk usually involves the application of a safety factor to a short-term exposure shown to produce no adverse effect. This dose is commonly known as the "no-observed-adverse-effect-level", or NOAEL. As in the case of chronic health risk assessment for non-carcinogens, the magnitude of the safety factor is determined by the nature and quality of the experimental data.

2.1.4 RISK CHARACTERIZATION

A quantitative measure of risk may be developed using the information derived from the exposure assessment and hazard evaluation components of the risk assessment process. Health risks resulting from long-term (chronic) and short-term (acute) exposure are characterized differently. So, too, are chronic health risks associated with exposures to carcinogens and non-carcinogens.

For chronic exposure to carcinogens, risk is characterized as excess lifetime probability of contracting cancer, and can be calculated by multiplying the risk per unit dose, the UCR, by the estimated lifetime daily dose. Since the development of a UCR is based on the assumption that carcinogens act via a no-threshold mechanism, some level of risk is associated with all finite exposures. Judgments must therefore be made as to what level of risk can be considered "acceptable". Frequently, public policy decision makers who must make this determination rely on precedents established as part of governmental regulatory action and/or comparisons to risks associated with other human activities.

For chronic exposure to non-carcinogens, the Acceptable Daily Intake (ADI) values developed in the hazard evaluation are compared to the predicted daily dose to determine whether or not the potential exists for effects in the exposed population. Acute exposures are evaluated in a similar manner by comparing the "safe" short-term dose to the predicted level of exposure.

2.2. LIMITATIONS AND UNCERTAINTIES ASSOCIATED WITH THE RISK ASSESSMENT PROCESS

Uncertainties associated with the process of risk assessment can be divided into two categories: (1) uncertainties inherent in the current state of knowledge, and (2) uncertainties resulting from deficiencies in available data. Uncertainties of the first type are primarily associated with hazard evaluation, and may result from:

- The extrapolation of toxicity data from animals to man,
- The extrapolation of high-dose/high-risk data to low-dose conditions, and
- The lack of data on biological interaction of toxic effects from combined exposures to several chemicals.

Uncertainties of the second type are typically associated with the exposure assessment, and may be reduced by:

- Use of measured rather than estimated values for emissions or environmental concentrations,
- Use of more sophisticated environmental fate and transport models, and
- Use of more detailed data on environmental conditions.

The existence of a degree of uncertainty should be noted in the interpretation of the results of any risk assessment. Despite these limitations, however, the risk assessment methodology provides a systematic approach to scientific evaluation of available data for assessment of public health risk. The use of appropriate data and assumptions in a risk assessment can provide a meaningful and useful measure of the potential for adverse health effects resulting from exposure to toxic substances. For this reason, risk assessment is often used as a basis for the development of regulatory policy.

2.3 APPLICATION OF RISK ASSESSMENT TO THE OWMC FACILITY

2.3.1 INTRODUCTION

The most highly ranked goal in the establishment of the proposed OWMC Hazardous Waste Treatment Facility is to minimize risks to human health. The second most highly ranked goal is to minimize risks to the environment. The principles of risk assessment have therefore been applied by OWMC to develop a quantitative estimate of the potential human health and environmental risks associated with facility operations. The information derived from the analysis will be used to evaluate the suitability of the proposed site and to identify elements of the facility design and operation which could be modified if necessary to reduce risks to human health and the environment. The results of the risk assessment will also provide a basis for the development of management strategies, including monitoring programs, to ensure that facility operations will not pose unacceptable human health and environmental risks.

The general approach taken in applying the principles of risk assessment in the current analysis are discussed in the following sections of this chapter.

2.3.2 EXPOSURE ASSESSMENT

The assessment of potential human exposures associated with releases from the proposed OWMC Facility consisted of the following four steps:

- Identification of sources of contaminant release to the environment;
- Characterization of the nature and magnitude of release from each source identified;
- Selection of human exposure pathways for analysis and the application of fate and transport models to predict contaminant concentrations in the environment; and
- Estimation of human dose by inhalation, ingestion or dermal absorption.

These four steps are briefly described in this chapter; more detailed discussions are provided in subsequent chapters of this report (Chapters 4, 5, 6, and 7) and in the technical appendices (Appendices C, D, E, and I).

2.3.2.1 Identification of Sources of Contaminant Release

The first step in the exposure assessment was the identification of sources of contaminant release that may be associated with the operation of the proposed OWMC Facility. In the analysis of potential releases to the environment, both normal operations and certain accident/upset conditions were considered.

Normal operations are distinguished by the routine emission of expected levels of contaminants from all potential sources of release. The routine emissions evaluated in the risk assessment were the following:

- Incinerator stack emissions;
- Stack emissions from the evaporation unit of the physical/chemical treatment plant (these emissions were calculated so as to include releases from makeup water sent to the incinerator scrubbing system);
- Leachate generated in the engineered landfill; and
- Fugitive emissions associated with
  - bulk unloading and transfer,
  - routine leaks and spills,
  - drum handling,
  - waste storage, and
  - physical/chemical treatment (emulsion breaking and dissolved air flotation)

These routine emissions were judged to be the most significant in terms of potential public health risks on the basis of a review of the proposed design and operation of the facility.

Contaminants may also be released to the environment as a result of upsets and accidents that occur at the facility. These events may produce increased emissions of chemicals from at least one point of release for a limited period of time. The on-site upsets and accidents examined in the risk assessment were the following:

- A fire in the tank farm storage area for organic wastes, resulting in potentially hazardous emissions to the atmosphere;

- Mixing of incompatible wastes in the physical/chemical building, resulting in the release of hydrogen sulfide gas;
- Failure of the incinerator stack flue gas scrubbing system, resulting in increased acid gas emissions;
- pH excursions during cyanide treatment in the physical/chemical plant, resulting in hydrogen cyanide emissions; and
- pH excursions during sulfide treatment in the physical/chemical plant, resulting in hydrogen sulfide emissions.

The public health risks associated with these events were previously evaluated in the Phase 4A Generic Risk Assessment (ENVIRON 1985), and are reexamined in this report. It should be noted that a detailed hazard analysis for the OWMC Facility is currently being conducted to identify additional on-site upset and accident scenarios, and that the hazards associated with any events identified as posing a potentially significant risk to public health will be examined as the results of that study become available.

Accidents involving vehicles transporting wastes, additives, or reagents to the proposed facility may also result in the release of potentially hazardous contaminants to the environment. Accidents which occur along local access routes to the facility have been judged to be of greatest concern due to the potential proximity of residences. Furthermore, it was determined as part of the Phase 4A Generic Risk Assessment that the risks posed by spills of hazardous wastes are significantly greater than those associated with spills of additives or reagents (ENVIRON 1985).

Two transportation accident scenarios have been evaluated in the risk assessment:

- A tank truck transporting liquid wastes to the OWMC Facility is involved in a collision on the local access route, leading to the release of chemicals onto the roadway.
- An equipment malfunction or transportation accident results in the release of liquid special wastes directly into a stream adjacent to the access road.

The public health risks associated with the first scenario were previously addressed in the Phase 4A Generic Risk Assessment (ENVIRON 1985).

2.3.2.2 Characterization of Releases

Potential contaminant releases from the proposed OWMC Facility have been characterized in terms of the following parameters:

- frequency and duration,
- chemical composition, and
- magnitude of release.

For routine emissions, it was assumed that contaminants would be released continuously for a period of 70 years. In the case of short-term releases associated with on-site and off-site upsets and accidents, the frequency and duration of potential exposures have been estimated based on engineering evaluations and assumptions regarding the operation of the proposed facility.

The composition of the various routine and non-routine releases from the OWMC Facility has been estimated based on both an understanding of the types of waste to be received and observations regarding emissions from similar facilities. The nature of the chemicals released from the facility are expected to vary from one emission source to another. For example, incinerator emissions may contain polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF), which can be formed during the combustion of special wastes. Routine fugitive releases would not be expected to involve appreciable quantities of PCDD/PCDF; the chemicals of greatest concern from these sources would be primarily volatile organic constituents of the hazardous wastes.

The magnitude of releases calculated in the risk assessment are based on the operation of a facility receiving 150,000 t/a of special waste. A sensitivity analysis was conducted to consider the risks associated with a 300,000 t/a facility. The sensitivity analysis was performed to evaluate the impacts associated with the possible future expansion of the proposed facility (Monenco 1987). The emission rates for the chemicals of concern were estimated for both the base case and the sensitivity analysis using empirical observations from comparable facilities (ORF 1987) and engineering calculations (Monenco 1987).

2.3.2.3 Prediction of Contaminant Concentrations in the Environment

Once the nature and magnitude of contaminant releases from the proposed facility were estimated, pathways for human exposure were selected, and appropriate models applied to predict chemical concentrations in the environment. In order to perform a detailed analysis of the risks associated with the operation of the OWMC special waste management facility, site-specific data, such as a knowledge of potential receptors and the physical characteristics of surrounding terrain, were required. This information was used to select the most significant exposure pathways for evaluation and to estimate contaminant concentrations in the relevant environmental media. For example, ponds and cisterns are used as a source of drinking water in the vicinity of the preferred West Lincoln Site. This potential human exposure pathway was therefore selected for evaluation in the risk assessment.

Exposure pathways can be characterized as either direct or indirect. For the purposes of this report, direct exposure is defined as exposure to chemicals present in the medium to which the emission originally occurs. For example, direct exposure to stack emissions occurs via inhalation of contaminated air; direct exposure to leachate from a landfill occurs via ingestion of contaminated ground water. Direct exposures to facility emissions can be divided into two categories: (1) long-term (chronic) exposures associated with normal facility operations; and (2) short-term (acute) exposures associated with facility upset conditions and accidents.

Indirect exposures result from the movement of chemicals from one environmental medium to another. For example, emissions from the incinerator stack may deposit onto the soil and eventually be taken up by a plant. Relevant indirect exposure pathways may include ingestion of contaminated soil or locally grown vegetables, consumption of beef and milk from locally raised cattle, the use of bodies of surface water as a supply of drinking water and/or dermal contact with contaminated dust and soil. The pathways selected for evaluation in the risk assessment are discussed in Chapter 5 (Routine Emissions to the Atmosphere), Chapter 6 (Releases of Leachate from the Engineered Landfill), Chapter 7 (Releases Associated with On-site Upsets and Accidents), and Chapter 8 (Releases Associated with Off-site Transportation Accidents).

In order to estimate the magnitude of potential exposures, contaminant concentrations were estimated for the various environmental media associated with the pathways selected for evaluation. For example, to develop the information necessary to model human exposure from the ingestion of locally grown vegetables, several environmental processes were considered, including atmospheric dispersion of chemicals released from the incinerator stack, wet and dry deposition rates onto the soil and the exposed surface of the plant, losses of the chemical from the soil, rate of chemical uptake from the soil, accumulation of contaminants within the plant, and the length of the growing season.

2.3.2.4 Estimation of Human Dose

The final step of the exposure assessment was the calculation of the human dose associated with the various facility emissions. Contaminant doses to members of the potentially exposed community were estimated from the predicted chemical concentrations in the environmental media using standard dietary and metabolic assumptions.

The level of exposure to contaminants released from the OWMC Facility is dependent upon an individual's location and behavior. For example, contaminants emitted from the incinerator stack will be dispersed by atmospheric mixing and turbulence; therefore, chemical concentrations in air will vary according to the distance from the stack. A person located at the point of highest chemical concentrations is likely to receive a higher inhalation dose than someone situated elsewhere in the site vicinity. Furthermore, higher doses are predicted the longer a person is exposed to emissions from the facility. Clearly, individuals at different locations in the site vicinity for different periods of time would not be expected to receive the same dose of a chemical emitted from the incinerator stack.

It is not usually possible, however, to estimate the likely level of exposure for different individuals in the vicinity of the OWMC Facility due to uncertainties in both current and future behavior patterns. Therefore, assumptions which are likely to lead to an overestimate in the level of human exposure have been made in the risk assessment. For example, for exposures via inhalation, all dose estimates for all sources of emission have been based on a person located permanently at the point of highest ground-level contaminant concentrations. Other conservative assumptions with respect to the calculation of human dose are summarized in Chapters 5 through 8.

2.3.3 HAZARD EVALUATION

The hazard evaluation component of the risk assessment involved an analysis of potential human health effects associated with exposures to the contaminants that may be released during operation of the proposed OWMC Facility. For contaminants that may be expected in routine emissions from the facility, the hazards associated with chronic exposures were considered. For those chemicals which may be released in the event of an upset or accident related to facility operations, the potential health effects corresponding to acute exposures were examined.

For chronic exposures to suspected carcinogens, Unit Cancer Risk (UCR) values have been taken from assessments prepared by the United States Environmental Protection Agency's Carcinogen Assessment Group (CAG 1985). For chronic exposures to chemicals not believed to have the potential to produce carcinogenic effects, Acceptable Daily Intake (ADI) values published by various program offices of the United States Environmental Protection Agency (USEPA 1986) were used in most cases. In instances where ADI values were not available, they were calculated by ENVIRON using data derived from laboratory animal experiments.

Acute safe exposure limits (ASELs) have been developed to evaluate the risks associated with short-term exposures. ASEL values were calculated based on the application of a safety factor to acute no-observed-effect-levels (NOELs). For some upset and accident events, e.g., the tank farm fire, short-term exposures were predicted to exceed the ASEL under certain conditions. In these cases, exposures were evaluated in terms of short-term public limits (STPLs) which are based on an acute no-observed-adverse-effect-level (NOAEL). Comparisons to the STPL provide a clearer understanding of the types of effects that might be expected if exposures greater than the ASEL are predicted.

Detailed toxicity profiles for most of the chemicals considered in the current analysis were compiled in conjunction with the Phase 4A Generic Risk Assessment (ENVIRON 1985). Although new toxicity profiles have not been generated in Phase 4B, ADI and UCR values have been reviewed and updated to reflect more recently available data. These updated ADI and UCR values are presented in Chapter 9. In addition, as part of the Phase 4B risk assessment, ENVIRON has conducted a more detailed review of the toxicity of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). This study was undertaken in response the results of the Phase 4A generic risk assessment, which indicated that PCDDs/PCDFs are a major contributor to the total cancer risk associated with the operation of the proposed facility. The results of ENVIRON's review are outlined in Chapter 9 and discussed in detail in Appendices G and H.

2.3.4 RISK CHARACTERIZATION

Risk characterization of the releases from the OWMC Facility was undertaken separately for (1) chronic exposure to carcinogens; (2) chronic exposure to non-carcinogens; and (3) acute exposures. The approaches used to evaluate these three types of exposures are described in detail in Chapter 10. In making the distinction between carcinogens and non-carcinogens, consideration was given to the particular route of exposure, since some chemicals, e.g., formaldehyde, may be carcinogenic by inhalation and not by ingestion or dermal absorption.

2.3.4.1 Chronic Exposure to Carcinogens

Cancer risks associated with exposures to individual carcinogens were calculated by multiplying the estimated daily human dose by the Unit Cancer Risk (UCR). It was assumed in the assessment that the risks corresponding to exposures to individual carcinogens could be added to provide an understanding of total cancer risk associated with facility emissions. It is recognized that this assumption can lead to an overestimate or underestimate of risk if antagonistic or synergistic (respectively) interactions occur. However, the data with which to predict how specific mixtures of chemicals will interact are usually not available, and therefore a procedure for assessing total risk was required. In Canada, there is no widely accepted procedure for evaluating the total cancer risk associated with exposures to complex chemical mixtures; thus, an additive model that has been recommended by the United States Environmental Protection Agency (USEPA 1985) was applied.

Since carcinogens are typically evaluated using a no-threshold model, some degree of risk is associated with all finite dose levels, i.e., it is not possible to assert that there will be no cancer risk from chronic exposure to a carcinogen. Thus, it is necessary for individuals, communities, and regulatory bodies to make judgments as to the level of incremental cancer risk that can be considered "insignificant" or "acceptable".

In judging the "acceptability" of cancer risks associated with environmental exposures, it is often useful to distinguish between "individual risk" and "total population risk". "Individual risk" is usually defined as an estimate of the increased lifetime cancer risk to a person located permanently at the point of greatest contamination. This measure provides an understanding of the largest predicted risk to any one individual, but not of the size of the population that may experience a given level of risk. "Total population risk" describes the aggregate risk to the exposed population as a whole, but does not provide an indication of the risks to the most highly exposed individuals.

For example, consider a situation where total exposures to a given chemical correspond to an incremental cancer risk of one-in-one-thousand (1×10^{-3}). If one thousand ($1 \times 10^{+3}$) people are located in this area, the "total population risk" could be estimated as one additional cancer ($1 \times 10^{-3} \times 1 \times 10^{+3} = 1$). If 10 thousand people were located in the area, the "total population risk" would increase to 10 additional cancers, although the "individual risk" would remain at 1×10^{-3}. In the Phase 4B risk assessment, the results of the analysis have been evaluated with respect to potential increases in "individual risk", which is generally a more stringent basis for sparsely populated areas. This approach is consistent with OWMC's objective to ensure that no one individual will be subjected to an "unacceptable" level of risk.

In the Phase 4A Generic Risk Assessment, a one in one million chance of an individual contracting cancer as a result of lifetime exposure to emissions from the proposed OWMC facility was considered to represent a conservative benchmark of "acceptability". Partially in response to comments from Phase 4A reviewers, the approach taken in Phase 4B is to provide a context for evaluating the results of the analysis, rather than to suggest that one level of risk should be defined as "acceptable". This approach is consistent with the fact that a variety of factors, including the conservativeness of the risk projections, the size of the population potentially exposed, and the nature of the project under consideration, can impact the level of risk that an individual or regulatory body might consider "acceptable".

2.3.4.2 Chronic Exposure to Non-Carcinogens

As discussed in Section 2.3.3, carcinogens and non-carcinogens are believed to act through different mechanisms. Therefore, non-carcinogenic risks were evaluated differently than those associated with exposure to carcinogens in the risk assessment. Chronic exposures to individual non-carcinogens were examined by comparing the estimated Acceptable Daily Intake (ADI) value with the calculated daily dose to determine an Adjusted Margin of Safety (AMOS). The AMOS values for individual chemicals were then aggregated for each organ of the body to allow for an estimation of the daily burden placed on an organ due to exposure to a mixture of non-carcinogenic substances.

The aggregation methodology used in the risk assessment is consistent with guidelines proposed by the United States Environmental Protection Agency (USEPA 1985). In the absence of data to the contrary, the USEPA has suggested that risks associated with exposures to mixtures of chemicals be estimated as though their effects were additive. This approach is considered to provide a reasonable estimate of the total risk associated with exposures to individual non-carcinogens for the purposes of the risk assessment. It should be noted, however, that detailed information on potential interactions between chemicals is required to rigorously evaluate the risks associated with mixtures. As noted in Section 2.3.4.1, these data are not available for the multi-component mixtures considered in the risk assessment.

In assessing the results of the analysis of chronic exposures to non-carcinogens, an aggregated AMOS value equal to or greater than one was considered to represent a situation where the risk to public health is very small. In such cases, the predicted level of exposure is below that considered to be sufficient to cause an adverse health effect. An aggregated AMOS less than one indicates that the predicted exposure may be significant, and could lead to human health effects.

2.3.4.3 Acute Exposures

For acute exposures, the characterization of risk was conducted by calculating an Adjusted Margin of Safety (AMOS) in a manner similar to that for chronic exposures to non-carcinogens. In this case, however, an Acute Safe Exposure Level (ASEL), which is based on an acute no-observed-effect-level (NOEL) to which a safety factor has been applied, was used for comparison to the human dose received over a short time period. For some exposures, depending upon the toxicological action of the chemical, it was more appropriate to make a comparison between a predicted concentration and an ASEL based on concentration rather than dose.

It should be emphasized that the use of an ASEL based on a NOEL in the characterization of acute risks represents a highly conservative approach. For acute exposures that occur relatively infrequently, a more conventional approach would involve the development of an acceptable level based on a no-observed-adverse-effect-level (NOAEL). The more conservative approach used to estimate AMOS values, however, was adopted as being consistent with the use of conservative assumptions throughout the risk assessment and OWMC's goal of minimizing risk to human health.

For acute exposures, an AMOS value less than one indicates that some effect may be observed. However, because of the use of the NOEL rather than the NOAEL in calculating an AMOS, these effects may not be adverse to human health. In such cases, the potential for adverse human health effects was evaluated using short-term-public-limit (STPL) values based on NOAELs to assess the potential acute health effects in cases where the AMOS values were less than one. A comparison with the STPL indicates whether a certain exposure would be expected to produce effects that are more significant than minor, irreversible irritation.

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CHAPTER 3

THE EXISTING ENVIRONMENT

3.1 INTRODUCTION

As discussed in Chapter 1, a generic risk assessment was conducted as part of the Phase 4A site selection activities (ENVIRON 1985). A primary objective of the Phase 4B Risk Assessment was to refine the Phase 4A analysis to better reflect the characteristics of the preferred site in West Lincoln Township. A variety of studies have therefore been commissioned by OWMC to gain an understanding of existing conditions in the vicinity of the preferred site (see, for example, Gartner Lee Associates 1987; M.M. Dillon 1987; and MDS Health Group 1986).

This chapter provides a brief description of the physical and land use characteristics of the area in the vicinity of the preferred site, and summarizes available data with respect to chemical concentrations in air, water, and soil near the site. This information was used in the human health risk assessment in (1) identifying and evaluating potential human exposure pathways, and (2) establishing the nature and magnitude of existing levels of contamination in the site vicinity.

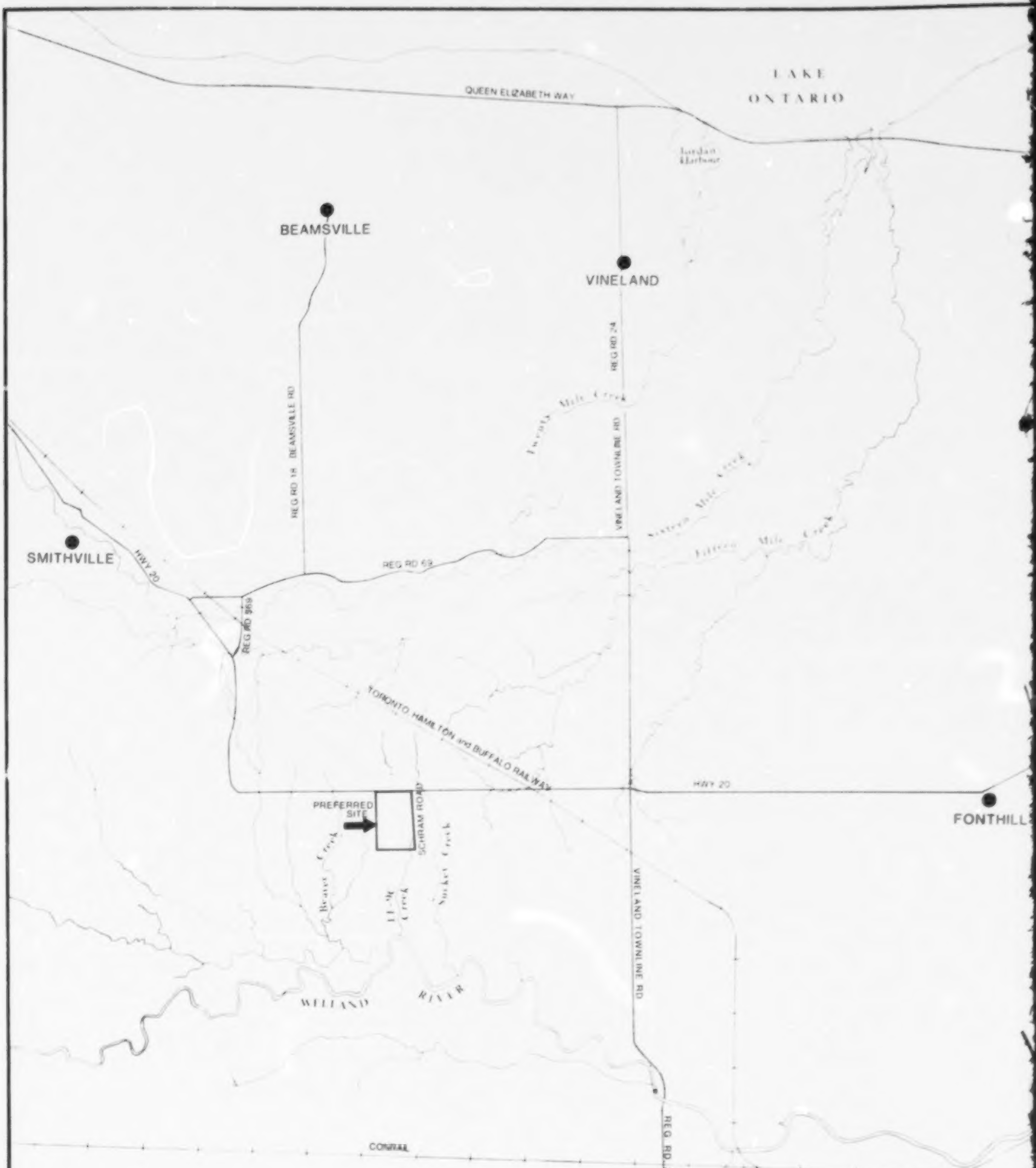
Information compiled with respect to the types of plants and animals living near the site served as the basis for selecting species for analysis in the environmental risk assessment. A summary of the types of livestock, crops, and wildlife in the vicinity of the preferred site is included in this chapter. A more complete description of wildlife and fish species that have been reported is presented in Appendix A.

3.2 DESCRIPTION OF THE SITE VICINITY

3.2.1 INTRODUCTION

The preferred site for the proposed OWMC Facility is located on the Niagara Peninsula in West Lincoln Township, Ontario. The site, shown in Figure 3, is 124 hectares in size, and is situated approximately equidistant between Lake Ontario and Lake Erie. As shown in Figure 4, the preferred access route to the proposed facility is comprised of the following roadways: Regional Road 24 South (Vineland Townline Road), King's Highway 20 West, and Schram Road South.

The area surrounding the preferred site is primarily agricultural, with approximately 95 percent of the land engaged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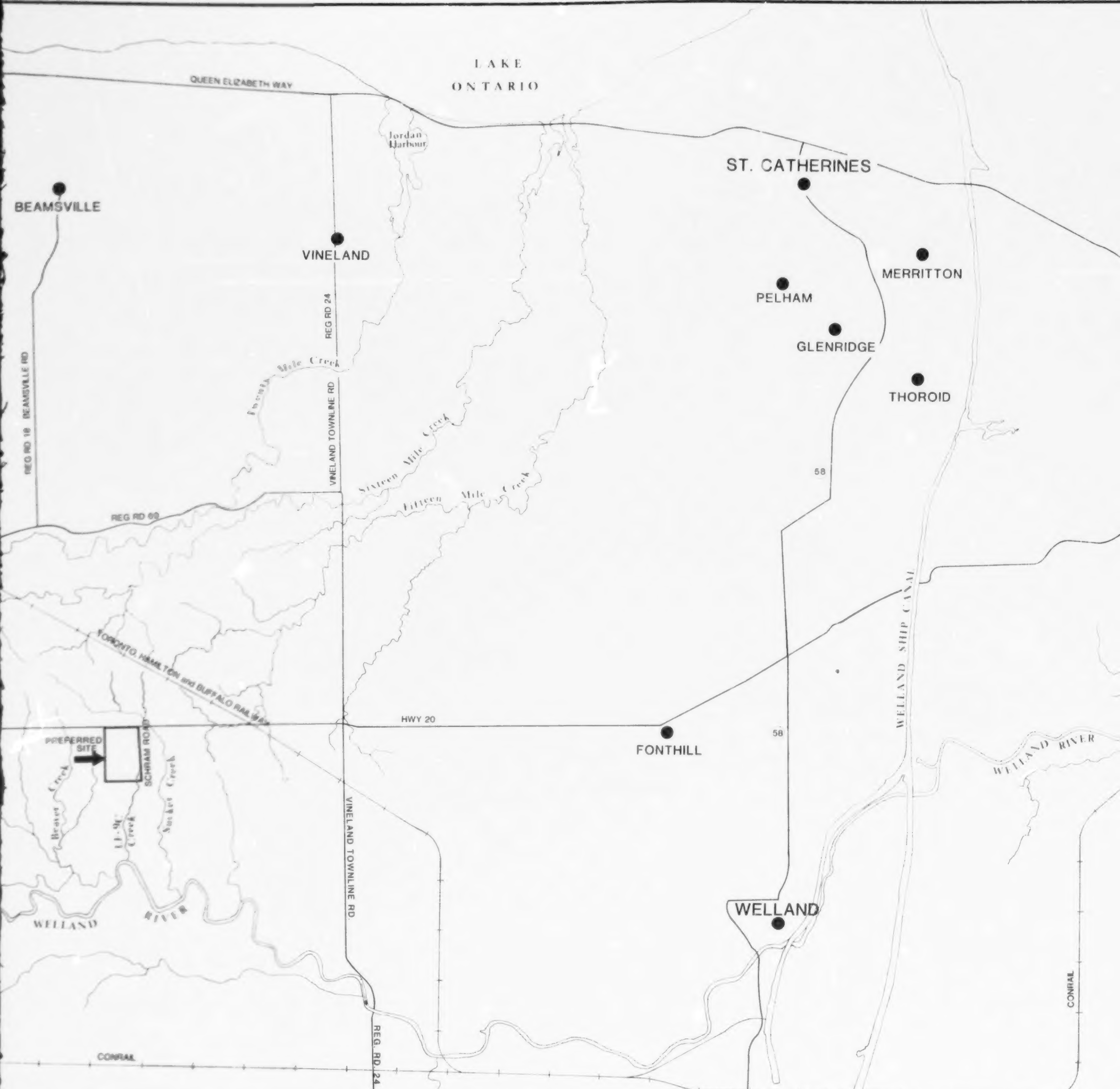
VINELAND TOWNLINE RD
Sixteen Mile Creek
Fifteen Mile Creek
SMITHVILLE
HWY 20
REG RD 69
REG RD 569
TORONTO HAMILTON and BUFFALO RAILWAY
HWY 20
FONTHILL
PREFERRED SITE
SCHRAM ROAD
Beaver Creek
Elm Creek
Sucker Creek
VINELAND TOWNLINE RD
WELLAND RIVER
CONRAIL
REG RD 24
3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QUEEN ELIZABETH WAY
Jordan Harbour
ST. CATHERINES
BEAMSVILLE
VINELAND
MERRITTON
PELHAM
GLENRIDGE
THOROID
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Twenty Mile Creek
REG RD 18 BEAMSVILLE RD
VINELAND TOWNLINE RD
Sixteen Mile Creek
Fifteen Mile Creek
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REG RD 69
WELLAND SHIP CANAL
TORONTO, HAMILTON and BUFFALO RAILWAY
HWY 20
FONTHILL
58
PREFERRED SITE
SCHRAM ROAD
Beaver Creek
Elsie Creek
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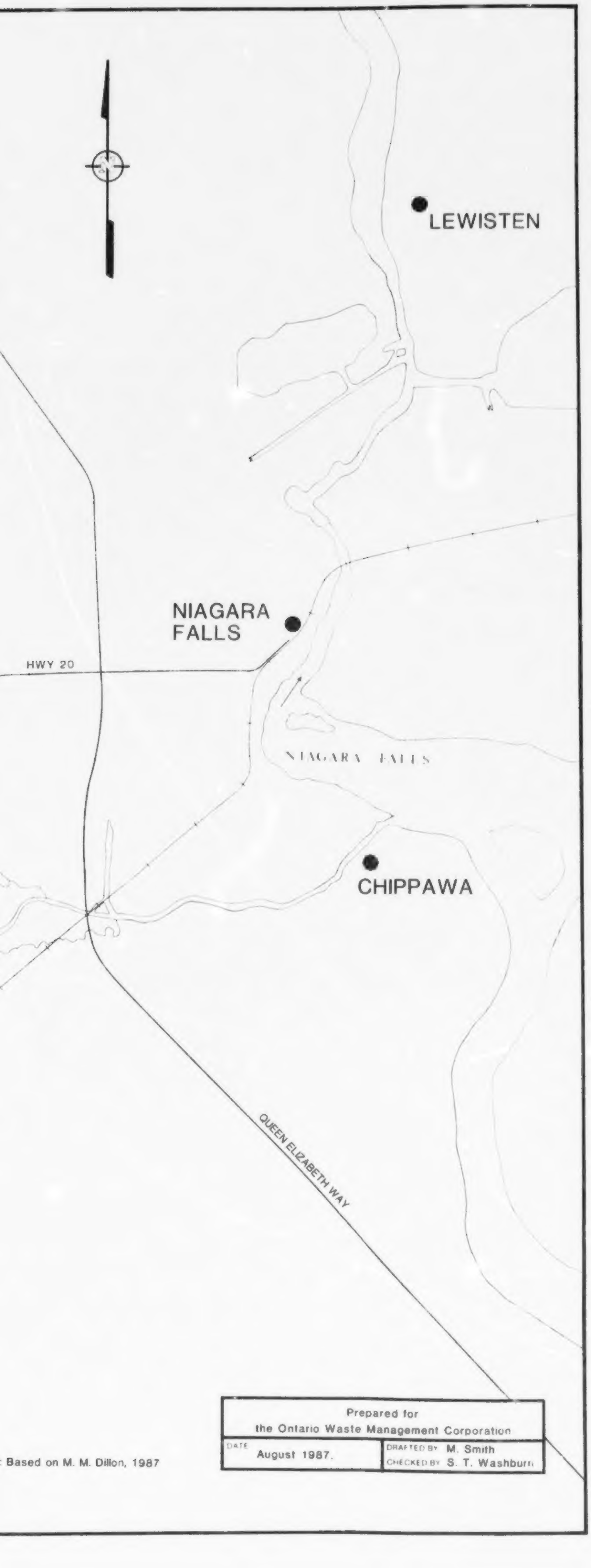

LEWISTEN
NIAGARA FALLS
HWY 20
NIAGARA FALLS
CHIPPAWA
QUEEN ELIZABETH WAY

Prepared for
the Ontario Waste Management Corporation

DATE August 1987.
DRAFTED BY M. Smith
CHECKED BY S. T. Washburn

Based on M. M. Dillon, 1987

Ontario Waste Management Corporation

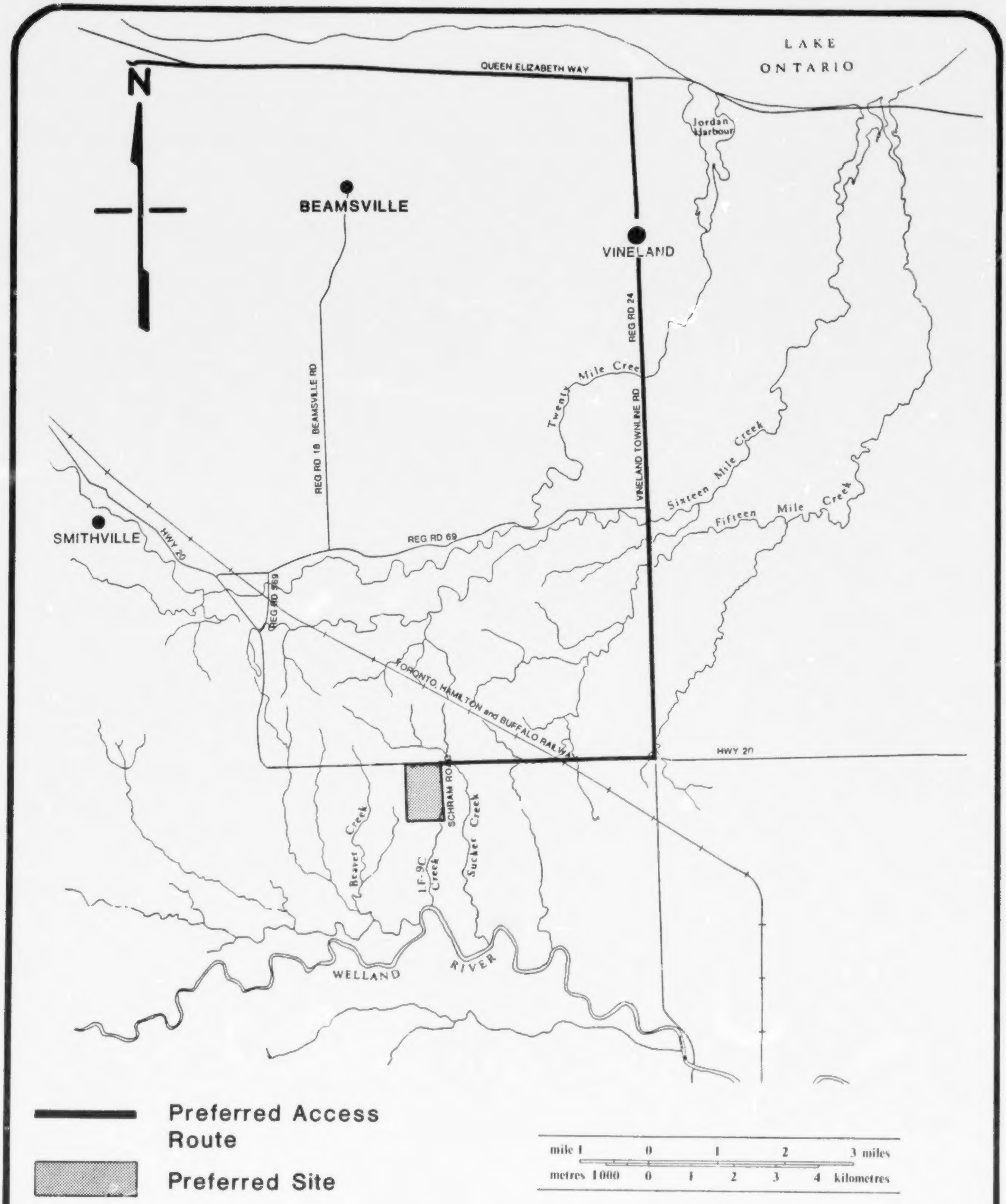
Consultant

ENVIRON

mile 1 0 1 2 3 miles
metres 1000 0 1 2 3 4 kilometres

The Preferred Site for the OWMC Facility

FIGURE 3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VINELAND TOWNLINE RD
Twenty Mile Creek
Sixteen Mile Creek
Fifteen Mile Creek
SMITHVILLE
HWY 20
REG RD 69
REG RD 569
TORONTO, HAMILTON and BUFFALO RAILWAY
HWY 20
SCHRAM ROAD
Beaver Creek
LF-9C Creek
Sucker Creek
WELLAND RIVER
Preferred Access Route
Preferred Site
mile 1 0 1 2 3 miles
metres 1000 0 1 2 3 4 kilometres

Based on M. M. Dillon, 1987

ENVIRON
Counsel in Health and Environmental Science

Preferred Access Route for the OWMC Facility

Figure 4

in farm-related activities (Ecologistics Ltd. 1987). Various bodies of surface water, including the Welland River as well as a number of small streams and ponds, are located in the vicinity of the preferred site (M.M. Dillon 1987). The nearest major urban center is the City of Welland, which is located along the Welland River about 15 km east of the proposed site. Other towns and hamlets near the preferred site include Vineland, Wellandport, Bismark, and Smithville. Approximately 75 residences are located within 2 km of the preferred site (OWMC 1987).

3.2.2 AGRICULTURE

West Lincoln Township, the site of the proposed facility, is one of the most important areas for livestock production and mixed farming in the Niagara Region. Approximately 75 percent of the dairy cows, 40 percent of the pigs, and 35 percent of the poultry in the Niagara Region are raised on farms in West Lincoln Township (OWMC 1986). A breakdown of the types of livestock found in the local community (an area within approximately 5 km of the preferred site) is presented in Table 3-1.

The nature of the soil near the preferred site limits the types of crops that can be grown. The surface soil, predominantly clay loam, is noted for having poor drainage

TABLE 3-1

LIVESTOCK IN THE LOCAL COMMUNITY[1]

	Total
Dairy Cows	1 173
Dairy Cows (under 1 year)	387
Replacement Heifers	407
TOTAL DAIRY CATTLE	1 967
Beef Cows	264
Beef Calves (under 1 year)	135
Replacement Heifers (older than 1 year)	47
Feeder Cattle	1 929
Bulls	10
TOTAL BEEF CATTLE	2 385
Sows	204
Boars	11
Replacement Gilts (young sows)	32
Pigs (under 2 months)	735
Feeder Pigs (older than 2 months)	4 112
TOTAL PIGS	5 094
Layer Hens	153 861
Broilers	513 156
Turkeys	72 900
Other Poultry	113 397
TOTAL POULTRY	853 314
Horses	63
Other	1 964

---

[1] An area within a radius of approximately 5 km from the Preferred site.

Source: Ecologistics Ltd. 1987.

characteristics and for for being unable to support fruit trees (OWMC 1986). As shown in Table 3-2, the crops of major importance in and around the site are primarily grains and include hay, corn, winter wheat, soybeans, barley, and oats. A variety of fruits and vegetables, including tomatoes, carrots and cabbage, are also grown in areas that may be impacted by facility emissions.

3.2.3 WATER RESOURCES

Various bodies of surface water, including a number of creeks, small ponds and the Welland River, are located in the site vicinity. The site drains to the south towards the Welland River via three creeks: Beaver Creek, Sucker Creek, and an unnamed stream (referred to as 9C Creek in this report). North of the site, Fifteen Mile Creek, Sixteen Mile Creek and Twenty Mile Creek drain toward Lake Ontario. Figure 3 shows the location of these water bodies in relation to the preferred site.

The Welland River, which is located approximately 2 km from the preferred site, is the longest river in the Niagara Peninsula. It travels southeastward from the Mount Hope area to the Niagara River (Chippawa Power Canal) and has a drainage basin which encompasses approximately 880 km^2 (M.M. Dillon 1987). Although the Welland River is not used as a potable water source, it is used to water livestock (M.M. Dillon 1987).

TABLE 3-2

CROPPING PATTERNS IN THE LOCAL COMMUNITY[1]

	Hectares
Corn	1 530
Barley	456
Soybeans	226
Wheat	568
Hay	2 378
Oats	253
Fruit Trees	22
Mixed Grain	178
Other	122
Total	5 733

---

[1] An area within a radius of approximately 5 km from the site center. Includes only the area covered by farm interviews.

Source: Ecologistics Ltd. 1987.

Beaver Creek, 9C Creek and Sucker Creek are located within the Welland River Drainage Basin. 9C Creek flows through the site and receives 70 percent of the site drainage, while Beaver Creek and Sucker Creek receive 26 percent and four percent, respectively (OWMC 1986). 9C Creek is an intermittent stream which flows during the winter and spring, but is dry during the summer. Farmers in the site area have reported that these creeks have been used to water livestock although they are not used as a potable water supply (M.M. Dillon 1987).

The Lake Ontario Drainage Basin, located north of the site, includes Fifteen Mile Creek, Sixteen Mile Creek and Twenty Mile Creek. Fifteen Mile and Sixteen Mile Creeks drain land about 2 km north of the site, and Twenty Mile Creek drains land 4 km to the north (M.M. Dillon 1987). As shown in Figure 4, the local access route crosses each of these streams.

Numerous ponds are located near the preferred site. Many of these ponds provide water for cattle and turkeys during the summer months, and some ponds are also used by people living in the area as a source of potable water (Gartner Lee 1987).

The most important source of drinking water north of the preferred site is a bedrock aquifer which flows beneath the site toward the Welland River (Gartner Lee 1987). Cisterns, which are replenished by rain water, and overburden wells, which collect shallow ground water, also represent supplies of potable water north of the site. In the south, most of the homes in the site area use cisterns as the primary source of both potable and non-potable water (OWMC 1986). Only four wells to the bedrock aquifer are currently located to the south of the preferred site between the site and the Welland River; only two of these wells are used as sources of drinking water (Gartner Lee 1987).

3.2.4 WILDLIFE

As shown in Appendix A, a total of 88 bird species, 18 mammalian species, and 12 reptile and amphibian species have been identified on the preferred site and site vicinity (Gartner Lee 1987b). Five rare species have been reported: eastern bluebird, orchard oriole, opossum, bobcat, and black rat snake.

A variety of fish species have been reported in the Welland River and in the streams near the preferred site (Gartner Lee 1987b). The most abundant fish species in the Welland River include brown bullhead, white crappie, and sheepshead. The streams (9C Creek, Beaver Creek, and Sucker Creek) to the south of the site support carp, mudminnow, brown bullhead, northern pike, and other species. A variety of sport fish, including coho salmon, rainbow trout, and brown trout have been found in the streams to the north of the site (Fifteen Mile, Sixteen Mile, and Twenty Mile Creeks).

Over 250 plant species have been identified on the preferred site (Gartner Lee 1987). Six of these species are considered rare in terms of their limited population size and distribution on a national, provincial or regional level. (As used here, the term "rare" does not mean that the species is endangered.) These six species are: big shellbark hickory, pin oak, blunt-leaved sandwort, tupelo, southern arrow-wood and spray aster.

3.2.5 IMPLICATIONS FOR THE RISK ASSESSMENT

The physical and land use characteristics of the site vicinity suggest that a number of potential exposure pathways may be significant in the human health risk assessment. For example, the agricultural nature of the area indicates that an evaluation of human exposures via ingestion of livestock and crops is important. The use of ponds and cisterns as a source of drinking water suggests that this pathway may also contribute to the risks associated with the operation of the proposed facility.

The site vicinity serves as the habitat for a variety of environmental species. Livestock, terrestrial wildlife, avian species, and numerous types of plant life live near the preferred site. In addition, the Welland River and various streams in the area support a range of aquatic species. It was therefore considered important to examine potential effects on a broad spectrum of species in the environmental risk assessment.

3.3 EXISTING LEVELS OF CONTAMINATION IN THE SITE VICINITY

3.3.1 INTRODUCTION

The concentrations of various chemicals in the air, soil, and surface water near the preferred site have been measured both by regulatory agencies (see M.M. Dillon 1987) and by consultants to OWMC (see, for example, Zenon 1987 and MDS 1986). These contaminant concentrations have been used in the risk assessment to help characterize background conditions in the vicinity of the preferred site. The existing levels of contamination have also been examined in assessing potential human health and environmental risks associated with the operation of the proposed OWMC Facility. For example, the estimation of risks associated with exposures to facility emissions have taken into account existing background concentrations where appropriate.

3.3.2 EXISTING CONTAMINANT CONCENTRATIONS IN AIR

Ambient air monitoring was conducted at the preferred West Lincoln site for a period of one year (June 1986-May 1987) to characterize existing air quality conditions (ORF 1987). Data were collected at an on-site monitoring station for various air quality parameters, including sulfur dioxide (SO_2), nitrogen oxides (NO_x), ozone (O_3), trace metals associated with particulate matter, and trace organic and inorganic constituents. Data for selected parameters are presented in Table 3-3.

Monitoring for volatile organics was also conducted near the preferred site using a mobile mass spectrometer unit. From a scan of over 70 chemicals, only benzene and toluene, at concentrations of 4 parts per billion (ppb) and 5 ppb, respectively, were detected (MDS 1986). It was concluded by MDS (1986) that the "area monitored was pristine with respect to high levels of volatile organics".

3.3.3 EXISTING CONTAMINANT CONCENTRATIONS IN SURFACE WATER

The Ontario Ministry of the Environment (OMOE) analyzed water taken from the Welland River between 1978 to 1986 for a variety of trace constituents including metals, phenols and cyanide (M.M. Dillon 1987). Except for phenols, concentrations of organic compounds were not measured.

TABLE 3-3

CONTAMINANT CONCENTRATIONS IN AMBIENT AIR AT THE PREFERRED SITE[1]

Pollutant	Concentration Range	"Average Maximum" Concentration[2]
Gaseous		
SO_2	12 ug/m^3 (seasonal mean)	--
NO_x	23 ug/m^3 (seasonal mean)	--
O_3	56 ug/m^3 (seasonal mean)	--
Air Samples		
Mercury	nd-20.5	6.4
PCBs	nd-0.073	0.032
Chlorobenzenes	nd-1.84	1.16
Chlorophenols	nd	nd
Pesticides	nd-0.14	0.096
Herbicides	nd-40	40
Dioxins	nd-0.00039	0.00039
Furans	nd-0.00022	0.00022
Polyaromatic Hydrocarbons	2.7-83.9	42.3
Total Suspended Particulate/ Fine Particulate Samples		
Arsenic	nd-2.6	0.83
Beryllium	nd	nd
Cadmium	nd-1.3	1.3
Chromium	nd-11.1	1.3
Copper	nd-25.6	17.4
Nickel	nd-5.5	1.7
Lead	nd-33.2	13.7
Zinc	nd-32.8	21.0

Source: ORF 1987.

[1] All concentrations in ng/m^3 unless noted.
1.0 nanogram (ng) = 1.0 x 10^{-9} gram;
1.0 microgram (ug) = 1 x 10^{-6} gram;
nd = not detected

[2] "Average Maximum": represents the average of the maximum concentrations detected during each of the four sampling periods.

The results of the sampling and analysis conducted for the Welland River from points upstream and downstream of the preferred site are given in Table 3-4. These data indicate that this section of the Welland River exceeds several OMOE drinking water criteria for a number of parameters, including turbidity, coliform, iron, copper, aluminum, phosphorus and ammonia. Thus, without considerable treatment, the Welland River could not be used as a drinking water source or for certain recreational purposes, such as swimming (M.M. Dillon 1987).

Water quality data for small streams and ponds near the site were not available.

3.3.4 BACKGROUND CONTAMINANT CONCENTRATIONS IN SOIL

Background concentrations of metals and organic compounds in soil taken from the vicinity of the preferred site are presented in Tables 3-5 and 3-6 (Zenon 1987). The following metals were detected: chromium (maximum concentration, 5.4 parts per million, or ppm), copper (maximum concentration, 1.24 ppm), lead (maximum concentration, 2.20 ppm), and zinc (maximum concentration, 5.96 ppm). The only organic compounds detected were acetone (at a maximum concentration of 114 parts per billion, or ppb) and naphthalene (at a maximum concentration of 120 ppb). Since acetone was used in the laboratory extraction

TABLE 3-4

WATER QUALITY DATA (1978-1986) FOR THE WELLAND RIVER
(Collected by the Ontario Ministry of the Environment)

	Concentrations (mg/l)[a]					
	Upstream of Preferred Site Sinclairville			Downstream of Preferred Site South of Welland Airport		
Parameters	Maximum Value	Mean Value	Minimum Value	Maximum Value	Mean Value	Minimum Value
Trace Constituents						
Aluminum	41.0	4.8	0.54	6.2[c]	5.05[c]	3.9[c]
Arsenic	0.013	0.003	0.001	0.001[b]	0.001[b]	0.001[b]
Cadmium	0.03	0.001	0.0001	--	--	--
Chromium	0.2	0.01	0.001	--	--	--
Copper	0.04	0.01	0.001	0.006[c]	0.005[c]	0.005[c]
Cyanide	0.11	0.06	0.01	--	--	--
Iron	48.0	5.1	0.7	16.0	4.3	0.9
Lead	0.043	0.009	0.003	0.004[c]	0.004[c]	0.004[c]
Manganese	0.7	0.29	0.02	0.53	0.128	0.03
Mercury (ug/l)	0.06	0.04	0.03	--	--	--
Nickel	0.11	0.007	0.001	0.008	0.008	0.008
Phenols (ug/l)	4.2	1.0	--	4.0	1.0	0.2
Zinc	0.12	0.017	0.003	0.031	0.022	0.014

---

[a] mg/l unless otherwise stated

[b] One sample analyzed

[c] Two samples analyzed

Source: M.M. Dillon 1987.

TABLE 3-5

SOIL CONCENTRATIONS OF METALS AT THE PREFERRED SITE

Metals	Detection Limit (ppm)	Maximum Soil Conc. (ppm)	Concentration Range (ppm)
Antimony	0.10	ND	–
Chromium	0.12	5.40	0 to 5.40
Chromium VI	0.40	ND	–
Copper	0.20	1.24	0 to 1.24
Lead	1.06	2.20	0 to 2.20
Mercury	0.04	ND	–
Selenium	0.02	ND	–
Silver	0.20	ND	–
Zinc	unknown	5.96	0.54 to 5.96

ND = Not Detected.

Source: Zenon 1987.

TABLE 3-6

SOIL CONCENTRATIONS OF VOLATILE ORGANIC COMPOUNDS NEAR THE PREFERRED SITE

Volatile Organic Compounds	Detection Limit (ppb)	Maximum Soil Conc. (ppb)	Concentration Range (ppb)
Acetone	2	114	27 to 114
Bromodichloromethane	5	ND	--
Bromoform	10	ND	--
Chlorobenzene	2	ND	--
Cresols	100	ND	--
Ethanol	20	ND	--
Methyl Ethyl Ketone	2	ND	--
Naphthalene	20	120	47 to 120
Nitrophenols	100	ND	--
Phenol	100	ND	--
Pyridine	2	ND	--
Toluene	2	ND	--
1,1,1-Trichloroethane	5	ND	--

ND = Not Detected.

Source: Zenon 1987

procedure, levels may be representative of laboratory contamination rather than soil contamination. Except for acetone, all of the aforementioned contaminants are present in concentrations that are indicative of relatively unpolluted soils.

3.3.5 Summary

Available data indicate that chemical concentrations in air and soil near the site are consistent with the rural, non-industrial nature of the area. Unexpectedly high concentrations of organics and metals have not been detected in air or soil. However, relatively high concentrations of a variety of metals and other pollutants have been detected in water taken from the Welland River. It is therefore possible that the river is unable to support certain sensitive aquatic species.

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CHAPTER 4

CHARACTERIZATION OF WASTES

4.1 THE OWMC WASTE PROFILE

The proposed OWMC Hazardous Waste Management Facility has been designed for the effective treatment and disposal of a broad spectrum of potentially hazardous wastes. To gain an understanding of the types and quantities of waste that may be received, OWMC commissioned various surveys of industries in the Province of Ontario (see, for example, OWMC 1983; OWMC 1984; OWMC 1985). The information compiled during these surveys has been used by OWMC and its engineering consultants to develop a profile of the estimated quantities and characteristics of the special wastes to be received at the proposed facility. This profile, known as the OWMC Waste Profile, consists of 14 general classes of potentially hazardous materials. The "typical" quantity associated with each class is presented in Table 4-1.

The classification of wastes allows for the identification of streams which are likely to be similar in certain

TABLE 4-1

THE OWMC WASTE PROFILE

	OWMC Waste Class	Typical Waste Quantity* (t/a)
1.	Organic Waste Sludges and Still Bottom	11 050
2.	Solvents and Organic Solutions	3 330
3.	Oil and Grease	880
4.	Oil and Water Mixtures	3 535
5.	Organic and Oily Residues	6 030
6.	Heavy Metal Solutions and Residuals	79 475
7.	Miscellaneous Chemicals and Products	3 100
8.	Paint and Organic Residuals	5 035
9.	Aqueous Solutions and Organics	1 785
10.	Anion Complexes	1 070
11.	Sludges and Inorganic Residuals	32 030
12.	Pesticides and Herbicide Wastes	175
13.	PCB Wastes	35
14.	Cleanup Residuals	2 470
	TOTAL	150 000

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*Rounded to the nearest 5 t/a.

Source: OWMC Waste Profile (Monenco 1987a).

characteristics, such as chemical composition. For example, waste streams in OWMC Waste Class 2, "Solvents and Organic Sludges" would be expected to contain appreciable quantities of volatile organics; OWMC Waste Class 6, "Heavy Metal Solutions and Residuals", would instead be predicted to consist primarily of inorganic material. This type of classification assists in the risk assessment process by grouping these wastes which could tend to pose similar hazards during treatment and disposal of the OWMC Facility.

For the purposes of the risk assessment, the waste classes listed in Table 4-1 can be categorized according to the type of treatment expected at the OWMC Facility:

- Organic Wastes, to be treated by the rotary kiln incinerator;
- Aqueous Organic Wastes, high water content streams to receive initial processing in the physical/chemical plant;
- Inorganic Wastes, to be received by the physical/chemical treatment plant;
- Inorganic Wastes, to be sent directly to solidification without pretreatment in the physical/chemical treatment plant.

The quantities of special waste associated with each of these treatment categories are listed in Table 4-2.

As shown in Table 4-2, inorganic wastes to be treated in the physical/chemical plant are projected to represent over

TABLE 4-2

THE TYPES OF WASTE PROJECTED TO BE RECEIVED AT THE OWMC FACILITY

	Typical Waste Quantities (t/a)[2]			
OWMC Waste Class[1]	Organic Wastes	Aqueous Organic Wastes	Inorganic Wastes Sent to the Physical/Chemical Plant	Inorganic Wastes Sent Directly to Solidification
1	9 580	1 470	–	–
2	3 330	–	–	–
3	880	–	–	–
4	3 535	–	–	–
5	3 790	2 240	–	–
6	–	–	66 055	13 420
7	860	115	2 125	–
8	4 745	290	–	–
9	600	1 185	–	–
10	–	–	1 070	–
11	–	–	12 680	19 350
12	175	–	–	–
13	35	–	–	–
14	2 470	–	–	–
TOTAL	30 000	5 300	81 930	32 770

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[1] Descriptions of the OWMC Waste Classes are provided in Table 4-1.

[2] The total quantity of inorganic residues to be solidified will be increased by the transfer of residues from certain treatment processes. For example, residues from the physical/chemical treatment plant and ash from the incineration process will be sent to the solidification unit prior to disposal in the engineered landfill.

Source: OWMC Waste Profile (Monenco 1987a).

half of the material to be received at the OWMC Facility. Approximately equal quantities of inorganic wastes to be sent directly to solidification and organic wastes to be incinerated are expected. Aqueous organic streams are predicted to represent only a small portion of the wastes predicted to be treated at the OWMC Facility.

The types of wastes projected to be received by the OWMC Facility can be described in greater detail using the University of California at Davis (UCD) waste classification system. In the UCD system, specific waste types are grouped according to chemical composition and physical state. In most cases, an OWMC Waste Class encompasses several UCD categories. For example, OWMC Waste Class 1 (Organic Waste Sludges and Still Bottoms) is composed of the following UCD classes:

UCD Waste Category	Description
247	Halogenated Still Bottoms
248	Non-Halogenated Still Bottoms
252	Solid or Sludge Tannery Waste
257	Phenolic Wastes
261	Polymeric Resins
263	Other Polymeric Wastes
446	Degreasing Sludge
523	Tank Bottom Sediments

Since a UCD category consists of only a portion of the material within an OWMC Waste Class, it generally provides a more detailed description of the nature of the various waste types. The UCD categories that comprise the organic, aqueous organic, physical/chemical inorganic, and solidification inorganic wastes are listed in Appendix B.

4.2 THE CANVIRO DATA BASE

Although the UCD classification system provides a description of the various types of wastes expected to be received at the OWMC Facility, it does not include the chemical-specific data required to conduct a quantitative risk assessment. Specifically, some knowledge of the identity and concentrations of individual chemicals in each type of waste is required to evaluate the potential hazard associated with the operation of the OWMC Facility. In the risk assessment, the data used to estimate the chemical composition of individual UCD categories were derived from a survey conducted for OWMC by CANVIRO Consultants, Ltd. (CANVIRO 1985). This survey was commissioned by OWMC to determine the types and quantities of special waste generated in the Province of Ontario. An additional objective in conducting the study was to develop information on the identity and concentrations of potentially hazardous constituents in the special wastes produced by a sample of 1000 manufacturers in Ontario.

For the purposes of this risk assessment, the data compiled by CANVIRO provide the most appropriate basis currently available for characterizing the composition of the wastes to be received at the OWMC Facility. For example, the information was provided by industrial facilities in the Province of Ontario and, therefore, corresponds to the specific types of special waste to be received at the facility. Furthermore, the CANVIRO data served as the basis for the proposed design of the OWMC Facility. It should also be noted that several of the organic chemicals reported in the largest quantities in the CANVIRO data base are also among the most prevalent constituents of incinerable wastes generated in the United States (MITRE 1983). There is, therefore, a degree of consistency between the composition of the wastes projected by CANVIRO to be received at the OWMC Facility and the composition of the wastes received at existing hazardous waste incinerators.

There are, however, several limitations associated with the use of the CANVIRO data base in the risk assessment. These limitations were discussed previously in the Phase 4A Generic Risk Assessment report (ENVIRON 1985), and are outlined below:

1. The database was developed from a partial survey of Ontario manufacturers that may not be fully representative of the range of industries in the Province.
2. The amount of quantitative waste composition data was, in some cases, limited. For example, concentrations of hazardous constituents identified in the CANVIRO database were not provided in certain cases.

3. For a few UCD classes, such as UCD 241 (pesticides), constituents that would be expected to be present were not reported, or were reported in small quantities.

4. The constituents of waste were frequently described in generic or descriptive terms (e.g. "oil and grease"). The identification of specific chemical compounds is necessary to rigorously evaluate the toxicity of a waste mixture.

5. The data with respect to specific organic contaminants in the aqueous organic and inorganic wastes are particularly sparse.

Given the limitations associated with the information provided by CANVIRO, assumptions were made in using the data to characterize special wastes for the purposes of the risk assessment. For example, the quantities of poorly defined but potentially hazardous contaminants have been represented by those hazardous constituents of the waste that are sufficiently well-defined for the purposes of risk assessment. The assumptions used in characterizing the special wastes are described in detail in Appendix B.

In addition, a supplementary data base (Franklin Associates 1985) was used to characterize the composition of "oil and grease," the generic contaminant identified in the greatest quantities in the CANVIRO data base. This data base was developed for the United States Environmental Protection Agency (USEPA) and includes information on the concentrations of specific hazardous constituents in used and waste oils. The Franklin data base and its use in the risk assessment are described in detail in Appendix B.

4.3 CHARACTERIZATION OF SPECIFIC WASTE TYPES

The OWMC Waste Profile (introduced in Section 4.1) has been used in the risk assessment to estimate the quantities of the various types of waste to be handled at the OWMC Facility. As discussed in Section 4.2, data provided by CANVIRO Consultants and Franklin Associates have served as the basis for defining the identity and concentration of specific chemicals within each type of waste.

The limitations associated with the use of the CANVIRO data base in defining the composition of special wastes in the risk assessment were summarized in Section 4.2. One problem is the identification of contaminants in only generic or descriptive terms. For example, contaminants in waste streams from some generators are referred to simply as "solvents." In a quantitative risk assessment, the identities of the specific chemicals of concern are necessary to evaluate the toxicity and potential human health effects in the exposed population. For example, both toluene and carbon tetrachloride are relatively common solvents. Both are known to be toxic, but carbon tetrachloride is considered to be a potential human carcinogen, whereas toluene is not. The risks associated with exposures to these two chemicals are therefore evaluated very differently (see Chapter 9). If a contaminant is identified only as a "solvent" in the CANVIRO data base, it is not clear whether the substance is toluene, carbon tetrachloride, one of numerous other chemicals used as solvents, or a mixture, making a quantitative assessment of risks impossible without the use of further assumptions.

In addition to the limitations of the CANVIRO data base, there are gaps in the current state of knowledge with respect to the toxicity of certain chemicals identified in the OWMC special wastes. For example, furfural is reported in some of the inorganic wastes, but sufficient toxicity data for evaluating chronic exposures to this chemical are not available.

An approach has been developed in the risk assessment to account for the total quantity of potentially hazardous contaminants reported in the CANVIRO data base, including those which are poorly defined (such as "solvents") and those for which sufficient toxicity data are currently unavailable (such as furfural). This approach is based on the selection of well defined chemicals for which adequate toxicity data are available to act as "surrogates" in representing the total quantity of potentially hazardous material in organic, aqueous organic, and inorganic wastes.

The methodologies used in the risk assessment to estimate the quantities of surrogate chemicals in each of the various treatment categories of waste are outlined in the remainder of this chapter. A detailed description of procedures used in developing these estimates is provided in Appendix B.

4.3.1 ORGANIC WASTES

Approximately 30 000 t/a of primarily organic wastes are expected to be received at the proposed OWMC Facility and treated in a rotary kiln incineration unit. It is necessary to characterize the organic constituents of these wastes to allow for an evaluation of the potential human health risks associated with releases from the incinerator stack. Organic substances present in the incinerable wastes are also expected to contribute significantly to fugitive emissions from the proposed facility.

The metal content of the organic wastes was not characterized in the risk assessment since metal emissions from the stack were based on measurements recorded at existing hazardous waste incineration facilities. This approach was adopted due to the availability of metal emission data from existing incinerators and the relative lack of data on specific metals in the organic wastes to be received at the OWMC Facility.

For the purpose of the risk assessment, the material in the organic wastes was classified as follows:

- Hazardous Liquid, defined in this study as liquids believed to have the potential to pose a risk to public health;
- Non-hazardous Liquid, defined in this study as liquids (such as water) not believed to pose a significant risk to public health;
- Solid, such as grit.

If it was not clear how best to classify a certain contaminant, it was conservatively assumed that the chemical was a "hazardous liquid". For example, contaminants identified as "tar and asphalt", "adhesives", and "plasticizers" in the CANVIRO data base were assumed to be entirely composed of hazardous liquid material. Details on the classification of the organic contaminants are presented in Appendix B.

A review of the CANVIRO data base indicates that, of the the 30 000 t/a of organic wastes to be received at the OWMC Facility, approximately 7 500 t/a can be classified as "hazardous liquid"; 12 000 t/a as "non-hazardous liquid"; and 10 500 t/a as "solid". Water represents approximately 60% of the material designated as "non-hazardous liquid." "Solid" wastes are primarily composed of grit (3 700 t/a), resins (1 800 t/a), leather and leather dust (1 600 t/a) and glass (800 t/a).

The composition of the "hazardous liquid" wastes has been represented in the risk assessment by a group of "surrogate" chemicals identified in the CANVIRO and Franklin data bases. The first step in the surrogate selection process was to create a listing of the quantities of all specific organic chemicals identified in these two data bases. The composition of contaminants identified in only generic terms, e.g., "chlorinated ethylenes", or "organic solvents", was estimated wherever feasible. For example, "chlorinated ethylenes" was assumed to consist of a 50/50 mixture of trichloroethylene and tetrachloroethylene, the two most common chlorinated ethylene compounds. "Organic solvents" was assumed to be comprised of the relative quantities of those solvents specifically identified in the CANVIRO data base.

Next, the list of specific constituents was analyzed to identify those chemicals projected to be received at the facility in the greatest quantities, and those which are likely to pose the greatest potential public health risks during handling and disposal. This was accomplished by ranking the constituents according to those parameters (toxicity, quantity, and potential for release to the environment) critical in assessing the risks associated with both incinerator stack emissions and fugitive releases. Based on the results of the rankings, a list of 25 surrogate chemicals and their concentrations in the organic wastes was developed. These chemicals are listed in Table 4-3.

TABLE 4-3

MASS FRACTIONS OF SURROGATE CHEMICALS IN THE LIQUID PORTION OF THE ORGANIC WASTES

Chemical	Quantity[1] (t/a)	Mass Fraction[2]
Acetone	653.2	3.4 E-02
Acrylonitrile	4.2	2.2 E-04
Benzene	47.1	2.4 E-03
Benzo(a)Anthracene	0.3	1.6 E-05
Benzo(a)Pyrene	0.1	6.5 E-06
Butadiene	0.4	1.9 E-05
Carbon tetrachloride	21.2	1.1 E-03
Chloroform	35.5	1.8 E-03
Ethanol	987.3	5.1 E-02
Ethylene glycol	1 244.9	6.4 E-02
Ethylene oxide	0.3	1.6 E-05
Formaldehyde	11.3	5.8 E-04
Hexachlorobenzene	0.2	9.2 E-06
Hydrazine	0.01	5.4 E-08
Methyl ethyl ketone	181.5	9.3 E-03
Methylene chloride	349.0	1.8 E-02
PCBs	45.1	2.3 E-03
Petroleum distillates	1 089.1	5.6 E-02
Pyridine	21.0	1.1 E-03
Tetrachloroethylene	528.6	2.7 E-02
Tetraethyl lead	0.1	4.3 E-06
Toluene	939.3	4.8 E-02
1,1,1-Trichloroethane	411.0	2.1 E-02
Trichloroethylene	469.1	2.4 E-02
Xylene	477.2	2.5 E-02
Total	7 517	0.386

---

[1] Projected quantity of the surrogate chemical in the liquid portion of the organic wastes (as based on the CANVIRO and Franklin data bases). See Appendix B for details on the approach used in the risk assessment.

[2] Mass fraction of surrogate chemical in the liquid wastes, i.e., mass of chemical divided by the mass of liquid waste. These values are expressed in exponential terms; for example, 3.4 E-02 is equal to 3.4 x 10^{-2}, or 0.034.

In the risk assessment, it was conservatively assumed that the "hazardous liquid" component of the organic wastes consists entirely of these 25 surrogate chemicals. In this way, those materials, such as "adhesives," that are poorly defined in the CANVIRO data base but which may pose a risk to human health were represented by the surrogate chemicals.

4.3.2 AQUEOUS ORGANIC WASTES

"Aqueous organic" wastes are those waste streams which have high water content and are therefore sent to the physical/chemical plant for initial processing. In the aqueous organic waste treatment system, organic material is separated from water in the emulsion breaking units and dissolved air flotation (DAF) and then redirected to the incinerator. The decanted water remains at the physical/chemical plant and is treated via carbon adsorption before being discharged to an evaporation unit.

It is expected that some chemicals initially present in the aqueous organic wastes may ultimately be emitted from the evaporator. It is therefore necessary to characterize the concentrations of specific organic chemicals in the aqueous organic wastes to allow for an assessment of releases from the evaporator stack and as well as fugitive emissions from the DAF and emulsion breaking units.

The approach used to characterize the aqueous organic wastes was similar to that outlined in the previous section for organic wastes. First, the wastes were classified into three groups:

- Hazardous, defined in this study as those substances believed to have the potential to pose a risk to public health.
- Non-hazardous, defined in this study as those substances (other than water) not believed to pose a significant risk to public health.
- Water

If it was not clear how to classify a certain contaminant in the aqueous organic wastes, it was conservatively assumed that the chemical was "hazardous." For example, it was assumed that the contaminant identified as "acetates" in the CANVIRO data base was "hazardous." Details on the classification of the constituents of aqueous organic wastes are presented in Appendix B.

A review of the CANVIRO data base indicates that, of the 5 300 t/a of aqueous organic wastes to be received at the OWMC Facility, approximately 155 t/a can be classified as "hazardous," 130 t/a as "non-hazardous", and 5 015 t/a as water. The greatest quantity of "non-hazardous" waste (other than water) is associated with that portion of the "oil and grease" not believed to pose a significant risk to public health during handling at the OWMC Facility.

Unlike the organic wastes, only a limited number of constituents were identified in the aqueous organic wastes characterized using the CANVIRO data base. Therefore, all of the well-defined hazardous contaminants identified were selected to represent the "hazardous organic liquid" portion of the waste. In this way, those substances (such as "acetates") that are not well defined in the CANVIRO data base are represented by specific hazardous chemicals. The quantities and concentrations of the surrogate chemicals in the aqueous organic wastes, as estimated using this approach, are presented in Table 4-4.

Surrogate concentrations in Table 4-4 are given on a water-free basis since some organics are expected to form a separate phase. The exclusion of water leads to larger mass fractions of organics in the waste, and thus higher estimates of the release of organic contaminants through volatilization. It should be noted, however, that some chemicals, such as phenol and formaldehyde, may tend to partition in the aqueous phase rather the organic phase. The predicted fugitive emission rates for such chemicals would therefore be overstated in the risk assessment. The water-free concentrations were used in the estimation of fugitive emissions from the OWMC Facility, as described in Appendix C.

TABLE 4-4

MASS FRACTIONS OF SURROGATE CHEMICALS IN THE NON-WATER PORTION OF THE AQUEOUS ORGANIC WASTES

Chemical	Quantity[1] (t/a)	Mass Fraction[2] (t/t)
Toluene	102.7	3.6 E-01
Phenols	37.6	1.3 E-01
Petroleum distillates	5.8	2.0 E-02
Xylene	1.8	6.3 E-03
1,1,1-Trichloroethane	0.9	3.1 E-03
Styrene	2.1	7.3 E-03
Acrylonitrile	2.1	7.3 E-03
Formaldehyde	0.79	2.7 E-03
Tetrachloroethylene	0.47	1.6 E-03
Trichlorotrifluoroethane	0.33	1.1 E-03
Trichloroethylene	0.27	9.4 E-04
Naphthalene	0.21	7.3 E-04
Butadiene	0.21	7.3 E-04
Dichlorodifluoromethane	0.17	5.9 E-04
Benzene	0.08	2.8 E-04
PCBs	0.01	3.5 E-05
Benzo(a)Anthracene	0.01	3.5 E-05
Benzo(a)Pyrene	0.004	1.4 E-05
Total	155.6	0.54

---

[1] Projected quantity of the surrogate chemical in the aqueous organic wastes (as based on the CANVIRO and Franklin data bases). See Appendix B for details on the approach used in the risk assessment.

[2] Mass fraction of surrogate chemical in the non-water portion of the aqueous organic wastes; i.e., mass of chemical divided by the mass of the non-water portion of the aqueous organic wastes. Values are expressed in exponential terms; for example, 3.6 E-01 is equal to 3.6 x 10^{-1}, or 0.36.

4.3.3 INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

The inorganic wastes to be received at the physical/chemical treatment plant are expected to contain large quantities of both alkaline and acid substances that will be neutralized in the proposed treatment system. A review of the CANVIRO data base shows that metals (iron, chromium, steel, etc.), and to a much lesser extent organics, will also be present in these wastes.

Liquid effluent remaining after the treatment of the inorganic wastes in the physical/chemical plant will be discharged to an evaporation unit or used in the scrubbing system for the rotary kiln incinerator. This effluent will consist primarily of water, but may be contaminated with trace levels of both organics and metals originally present in the inorganic wastes. Non-pumpable residues generated as a by-product of the treatment processes will be solidified and deposited in the engineered landfill. Therefore, metals and organics may be emitted from the evaporator and the incinerator scrubber, or be present in leachate produced in the OWMC landfill.

The inorganic wastes sent to the physical/chemical treatment plant have been classified in the risk assessment as follows:

- Hazardous Inorganic, defined in this study as those inorganics not expected to be neutralized in the physical/chemical plant and that have the potential to pose a risk to public health.
- Hazardous Organic, defined in this study as those organics that have the potential to pose a risk to public health.
- Non-hazardous, defined in this study as those chemicals that are expected to be neutralized in the physical/chemical plant or that are not believed to pose a significant risk to public health.

In cases where it was not clear how best to classify a certain contaminant, it was conservatively assumed that the chemical was potentially hazardous. For example, the contaminant identified as "anhydrides" in the CANVIRO data base was assumed to be a "hazardous organic". The classification procedures are detailed in Appendix B.

As in the characterization of aqueous organic wastes, the entire quantity of potentially hazardous material in the inorganic streams to be sent to the physical/chemical plant was represented by the surrogate chemicals identified in either the CANVIRO or Franklin data bases. In this way, the quantities of contaminants in the inorganic wastes that are poorly defined were taken into account in the risk assessment. The quantities of the organic and inorganic surrogate chemicals as estimated using this approach are shown in Tables 4-5 and 4-6, respectively.

TABLE 4-5

QUANTITIES OF ORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Chemical	Quantity[1] (t/a)
Organics	
Toluene	342.7
Phenols	104.4
Styrene	40.8
Acrylonitrile	40.8
Petroleum distillates	4.3
Cresols	2.1
Ethylene glycol	1.9
1,1,1-Trichloroethane	0.88
Xylene	0.83
Tetrachloroethylene	0.41
Trichlorotrifluoroethane	0.31
Trichloroethylene	0.21
Naphthalene	0.21
Dichlorodifluoromethane	0.16
Benzene	0.05
Benzo(a)Anthracene	0.01
PCBs	0.01
Benzo(a)Pyrene	0.005
Total	540.0

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[1] Projected quantity of the surrogate chemical in the physical/chemical inorganic waste streams (as based on the CANVIRO and Franklin data bases). See Appendix B for details on the approach used in the risk assessment.

TABLE 4-6

QUANTITIES OF INORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Chemical	Quantity[1] (t/a)
Inorganics	
Cyanide	22.9
Fluoride	0.07
Nitrate[2]	1.6
Metals	
Zinc	753.4
Chromium	193.5
Copper	153.7
Nickel	145.3
Silver	29.6
Cadmium	15.5
Lead	12.5
Arsenic	6.8
Antimony	6.7
Mercury	0.77
Barium	0.02
Total Metals	1 317.8

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[1] Projected quantity of the surrogate chemical in the physical/chemical inorganic waste streams (as based on the CANVIRO and Franklin data bases). See Appendix B for details on the approach used in the risk assessment.

[2] Using the CANVIRO data base, over 400 t/a of nitrates would be predicted to be received by the physical/chemical treatment plant. Since this material is a valuable commodity, the engineering consultants to OWMC have determined that it is likely that little nitrate would actually be treated by OWMC (Monenco 1987b).

4.3.4 INORGANIC WASTES SENT DIRECTLY TO SOLIDIFICATION

This category of waste consists of primarily inorganic materials to be treated in the solidification unit. The solidified residues from this unit will be deposited in the engineered landfill. It was therefore necessary to characterize the identity and concentrations of individual chemicals in the inorganic wastes to be sent directly to solidification to evaluate the risks associated with exposure to leachate that will be generated in the landfill.

The approach used to characterize the inorganic wastes to be sent directly to solidification is identical to that previously described in Section 4.3.3. The quantities of the surrogate organic and inorganic chemicals in the inorganic wastes estimated using this approach are presented in Tables 4-7 and 4-8, respectively.

It should be noted that residues from the physical/chemical plant and incinerator will also be sent to the solidification unit for treatment prior to placement in the engineered landfill. Estimates of the contaminant quantities in both of these types of residues are presented in Appendix C.

TABLE 4-7

QUANTITIES OF ORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES SENT DIRECTLY TO SOLIDIFICATION

Chemical	Quantity[1] (t/a)
Phenols	110.7
Formaldehyde	62.4
Toluene	58.3
Petroleum distillates	3.9
Xylene	0.75
1,1,1-Trichloroethane	0.53
Tetrachloroethylene	0.24
Trichlorotrifluoroethane	0.19
Cresols	0.15
Trichloroethylene	0.12
Naphthalene	0.12
Ethanol	0.10
Dichlorodifluoromethane	0.10
Benzene	0.04
PCBs	0.007
Benzo(a)Anthracene	0.006
Benzo(a)Pyrene	0.003
Total	237.0

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[1] Projected quantity of chemical in the solidification inorganic waste streams (as based on the CANVIRO and Franklin data bases). See Appendix B for details on the approach used in the risk assessment.

TABLE 4-8

QUANTITIES OF INORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES SENT DIRECTLY TO SOLIDIFICATION

Chemical	Quantity[1] (t/a)
Inorganics	
Cyanide	1.0
Fluoride	0.01
Nitrate	4.9
Metals	
Zinc	1 765.4
Lead	923.4
Copper	912.9
Chromium	304.4
Nickel	229.0
Cadmium	4.1
Mercury	2.6
Silver	0.98
Arsenic	0.08
Barium	0.05
Total Metals	4 143.0

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[1] Projected quantity of chemical in the solidification inorganic waste streams (as based on the CANVIRO and Franklin data bases). See Appendix B for details on the approach used in the risk assessment.

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CHAPTER 5

ROUTINE EMISSIONS TO THE ATMOSPHERE

5.1 INTRODUCTION

As discussed in Chapter 2, the exposure assessment component of the risk assessment for the OWMC Facility consisted of the following four steps:

- Identification of sources of contaminant release to the environment;
- Characterization of the nature and magnitude of release from each source identified;
- Selection of human exposure pathways for analysis and application of fate and transport models to predict contaminant concentrations in the environment; and
- Estimation of human dose by inhalation, ingestion, or dermal absorption.

These steps in evaluating the routine atmospheric releases from the OWMC Facility are discussed in this chapter.

5.2 SOURCES OF RELEASE

Normal operation of the proposed OWMC Facility is expected to result in the routine release of potentially hazardous contaminants to the atmosphere. The risk assessment included an evaluation of the following types of routine atmospheric emissions:

- Incinerator stack emissions;
- Evaporator stack emissions; and
- Fugitive emissions resulting from
    - bulk waste unloading and transfer,
    - routine leaks and spills,
    - drum handling,
    - waste storage, and
    - physical/chemical treatment processes.

The nature and magnitude of these potentially hazardous releases were estimated in the risk assessment using a combination of empirical data generated at comparable facilities (see, for example, ORF 1987 and Midwest Research Institute [MRI] 1984) and engineering calculations (see, for example, Monenco 1987). The general methods used to estimate routine atmospheric emission rates are discussed this chapter. Details on the approaches employed are presented in Appendix C.

5.2.1 INCINERATOR STACK

5.2.1.1 Introduction

A high temperature rotary kiln incinerator will be used at the OWMC Facility to treat organic wastes. Gases generated during the combustion process will be treated by an air pollution control system consisting of a spray dryer, an electrostatic precipitator, and a three stage wet gas scrubber. The flue gas from this system will be vented to the atmosphere via the incinerator stack and may include the following types of toxic constituents:

- Uncombusted residues of organic chemicals in the waste feed;
- Metals present in the waste feed;
- Acid gases generated by combustion; and
- Products of incomplete combustion.

The characteristics of each of these emissions are discussed in the following subsections.

5.2.1.2 Uncombusted Organic Residues

Residues of the organic feed to the OWMC incinerator are expected to be present in the stack emissions since absolutely complete combustion of wastes will not occur. Testing at existing hazardous waste incinerators indicates that, in practice, destruction and removal efficiencies (DREs) of approximately 99.99 percent can be achieved (MRI 1984). If a DRE of 99.99 percent is reached for a given organic chemical, then for every 100 kg of that chemical fed to the incinerator, 99.99 kg would be destroyed, and 0.01 kg would be emitted from the stack. An enhanced DRE is required by some regulatory bodies for certain chemicals; for example, both the Ontario Ministry of the Environment (OMOE) and the United States Environmental Protection Agency (USEPA) require a 99.9999 percent DRE for the incineration of polychlorinated biphenyls (PCBs). This higher DRE is accomplished through more severe incineration conditions, e.g., higher temperatures in the combustion zone.

In the risk assessment, the emission rate for uncombusted organic feed residues was based on an expected total organic carbon (TOC) concentration of 15 mg/Nm^3 in the stack gases. This value was provided by EEC/Von Roll, incinerator technology consultants to OWMC, as the expected TOC concentration during normal operation of the incinerator (ORF 1987). The guaranteed maximum half-hour TOC concentration guaranteed by EEC/Von Roll is 56 mg/Nm^3 (ORF 1987).

The identity and quantity of specific organic constituents comprising the TOC emissions was based on information derived primarily from the CANVIRO data base (CANVIRO 1985). Emission data from existing facilities was judged to be inappropriate for use in this risk analysis due to large variations in waste feed composition that may occur from one incinerator to another, as well as limitations in the data that are currently available.

As discussed in Chapter 4, the CANVIRO data base has been used in the risk assessment to select "surrogate" chemicals to represent the total quantity of potentially hazardous material to be fed to the incinerator. Details on the methodology used to predict incinerator stack emission rates for the chemicals of concern are presented in Appendix C. It should be noted that the approach used in the risk assessment results in incinerator stack emission rates corresponding to average destruction and removal efficiencies (DREs) of approximately 99.9 percent for all compounds other than PCBs, for which a DRE of 99.999 percent has been applied. The calculated emission rates for organic constituents of the feed are, therefore, considerably higher than would be calculated assuming the 99.99 percent DRE achieved for principal organic hazardous constituents at existing hazardous waste incinerators (see Monenco 1986). This conservative approach reflects the lack of complete characterization of emissions at existing facilities.

5.2.1.2 Metals

The special wastes to be treated in the OWMC incinerator will be primarily organic in nature. However, some inorganic substances, such as metals, are also expected to be constituents of these wastes. Metals which are present in the incinerator feed may evaporate in the rotary kiln; most will subsequently condense to form aerosols of submicron particles in the cooler components of the incineration system, such as the heat recovery boiler or the acid gas scrubber.

Studies conducted at existing facilities indicate that the amount of non-volatile metals emitted from the incinerator stack is likely to be small relative to the total quantity in the feed. For example, testing at the West German Biebeshiem waste incinerator showed that at least 95 percent of the iron, chromium, copper, nickel, lead, cadmium, zinc, and tin remained in solid residues generated by the incinerator (ORF 1987). The residues included in the slag produced in the rotary kiln, the boiler ash, and the salt and flyash collected in the air pollution control equipment. The small quantities of these non-volatile metals that are emitted from the incinerator stack are typically associated with flyash that escapes the particulate removal scrubbing system.

Some relatively volatile metals, especially mercury, may not condense readily and are therefore more likely to be emitted from the incinerator stack as vapors. Emissions of these volatile metals are more difficult to control since they are not retained as efficiently by particulate control devices. The testing conducted at the Biebeshiem facility, for example, suggested that approximately 50 percent of the mercury in the waste feed was emitted from the incinerator stack (ORF 1987).

The Ontario Research Foundation (ORF) has conducted a review of metal emission data from existing hazardous waste incineration facilities (ORF 1987). These data are presented in Appendix C. For the purposes of the risk assessment, the highest emission rate observed for each metal from any existing hazardous waste incinerator has been conservatively adopted in predicting releases from the proposed OWMC Facility. This approach was adopted due to limitations in the data on specific metals in the wastes to be incinerated at OWMC.

5.2.1.3 Acid Gases

A variety of acid gases, including hydrogen chloride (HCl), hydrogen fluoride (HF), sulfur dioxide (SO_2), and nitrogen oxides (NO_x), may be formed during the incineration of hazardous wastes. The extent to which these acid gases are generated and subsequently released from an incineration facility is dependent upon the composition of the waste feed.

the design and operation of the incinerator, and the effectiveness of the pollution control equipment. Expected emission rates for the proposed OWMC incinerator have been provided by EEC/Von Roll, the incinerator technology consultant to OWMC (Monenco 1986, 1987). These projections are presented in Appendix C and were used in the risk assessment.

5.2.1.4 Products of Incomplete Combustion

Products of incomplete combustion (PICs), such as polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF), may be formed during the incineration of chlorinated hazardous waste. These compounds, which by definition are not present in the waste feed, must be considered in evaluating the potential health risks associated with incinerator stack emissions. In the risk assessment, those PICs such as PCDD/PCDF that have been detected in the stack effluent from existing hazardous waste incinerators have been considered.

Data on the long-term emission of PCDD and PCDF from hazardous waste incinerators have been compiled and reviewed by the Ontario Research Foundation (ORF 1987). Of the facilities included in the review, Biebeshiem in West Germany and SAKAB in Sweden are most similar in design and operation to the facility proposed by OWMC. Therefore, data from these two incinerators were considered by ORF to best reflect potential PCDD/PCDF emissions from the OWMC Facility. Based on their review, ORF recommended the use of one particular set of PCDD/PCDF emission data collected at the Biebeshiem facility for use in the risk assessment (ORF 1987). These data, and the projected PCDD/PCDF emissions from the proposed OWMC incinerator, are presented in Appendix C.

Testing at existing hazardous waste incineration facilities for PICs other than PCDD/PCDF has been limited. The most complete data available have been collected at various types of hazardous waste incinerators in the United States (MRI 1984). Ideally, data generated at rotary kiln units similar to the OWMC Facility in design and operation would be used in projecting PIC emissions. However, since PIC emissions data specific to rotary kiln units are largely unavailable, the data compiled by MRI for a broader range of incinerator types were used in the risk assessment to estimate PIC emissions for the OWMC Facility. The PIC emission data from hazardous waste incinerators reported by MRI are summarized in Appendix C along with projections developed for use in the risk assessment.

5.2.2 EVAPORATOR UNIT

The physical/chemical plant will consist of two separate processing systems for the treatment of inorganic and aqueous organic wastes. Practically all potentially hazardous chemicals in these wastes will be either destroyed or stabilized within the physical/chemical plant or separated for combustion in the rotary kiln incinerator. Liquid effluent composed mainly of water will be reused within the facility (e.g., make-up water to the incinerator scrubber) or will be discharged from the physical/chemical plant to an evaporation unit and subsequently vented to the atmosphere.

Small quantities of waste constituents may be present in the liquid effluent received by the evaporation unit and will, therefore, be emitted from the evaporator stack. In addition, some trace organics in the liquid effluent used in the incinerator scrubber may be emitted through the incinerator stack. Both sources were treated together as evaporator emissions for the purposes of the risk assessment. Any potentially hazardous emissions from the evaporator are expected to consist primarily of organic chemicals, although it is possible that small quantities of metals may also be entrained in the steam.

The first step in characterizing evaporator emissions was to estimate contaminant concentrations in the special wastes treated in the physical/chemical plant. As discussed in Chapter 4, information provided in the CANVIRO (1985) data base served as the basis for estimating contaminant concentrations in the special wastes. However, a supplementary data base developed by Franklin Associates (1985) was used to characterize the hazardous constituents of "oil and grease". "Oil and grease" is predicted by the CANVIRO data base to be the contaminant of greatest quantity in the aqueous organic wastes. Details on the estimation of contaminant concentrations in the waste to be received at the physical/chemical plant are presented in Appendices B and C.

After chemical concentrations in the wastes were estimated, contaminant destruction or removal in the various physical/chemical treatment processes was predicted. As previously noted, practically all of the potentially hazardous waste received by the physical/chemical plant will be destroyed, stabilized, or separated and sent either to the incinerator or the solidification unit. Furthermore, liquid effluent from the physical/chemical plant will be polished with reverse osmosis and activated carbon. The concentrations of specific chemicals remaining in the polished liquid stream to the evaporator were estimated by the engineering consultants to OWMC for the purposes of the risk assessment (Monenco 1987).

The final step in the analysis was to calculate routine evaporator stack releases based on the predicted chemical concentrations in the discharge to the evaporator unit. For organic chemicals, the average contaminant emission rate was based on the flow of the specific waste stream and the contaminant level after polishing. It was assumed that all organics sent to the evaporator would be vaporized and emitted. For inorganic contaminants, it was assumed that releases would result primarily from the entrainment of water droplets in the evaporator plume (Monenco 1987).

Details with respect to the methods used to predict evaporator stack emissions are included in Appendix C.

5.2.3 FUGITIVE RELEASES

Fugitive contaminant emissions to the atmosphere may be generated at various locations at the proposed OWMC Facility. In the current risk assessment, releases associated with the following operations were evaluated:

- Bulk waste unloading and transfer. Although wastes may be unloaded in a variety of ways at the proposed facility, unloading into underground tanks was identified as having the most significant emissions. This mode of waste unloading was therefore analyzed in the risk assessment. These unloading tanks will be equipped with covers or doors and an extraction fan connected to a vent exhaust system. Exhaust from the fans could be routed to the incinerator, or treated by an emission control system, such as a scrubber/adsorber before being released to the atmosphere.
- Routine leaks and spills. Routine leaks and spills may be expected at any industrial facility. In the risk assessment, routine spills during waste transfer operations, and liquid and vapor leaks from valves, pumps, and other equipment, have been examined.

- Drum handling. Both organic and inorganic chemicals may be released to the atmosphere as the result of various drum handling operations at the proposed OWMC Facility. For example, fugitive vapors will be generated during the handling of open drums. In addition, fugitive releases will be produced during the heating of drums of high viscosity wastes. Organic vapors from both operations will be collected and exhausted to the atmosphere through an emission control system, such as an activated carbon adsorber.

- Waste storage. Waste storage tanks will be used for storage, blending, and pretreatment of pumpable wastes. Fugitive emissions will result from the displacement of vapors from the storage tanks as they are being filled. During normal operating conditions, hydrocarbon emissions from organic waste storage will be routed directly to the incinerator. In the event that the incinerator is out of service, however, gases will be vented to the atmosphere through an activated carbon adsorber. Fugitive emissions associated with inorganic waste storage are also expected.

- Physical/chemical treatment. Certain aqueous organic and inorganic wastes will be treated in the physical/chemical treatment plant. Organic fugitive emissions are expected from the dissolved air flotation (DAF) and emulsion breaking units, which separate hydrocarbons from water. These releases will be exhausted to the atmosphere through an emission control system, such as activated carbon adsorber. Inorganic emissions may result from oxidation/reduction reactions for the treatment of cyanides.

Organic fugitive emission calculations provided by the engineering consultants (Monenco 1987) for use in the risk assessment were developed on the basis of a single component system, i.e., an individual chemical. In order to extend these single component system estimates to the multiple component system found in a waste mixture, Raoult's Law for ideal vapor-liquid equilibria was used to approximate the behavior of hazardous wastes mixtures.

The specific methodologies employed in calculating fugitive emissions from the various sources at the proposed OWMC Facility are presented in Appendix C.

5.3 PATHWAYS FOR HUMAN EXPOSURE

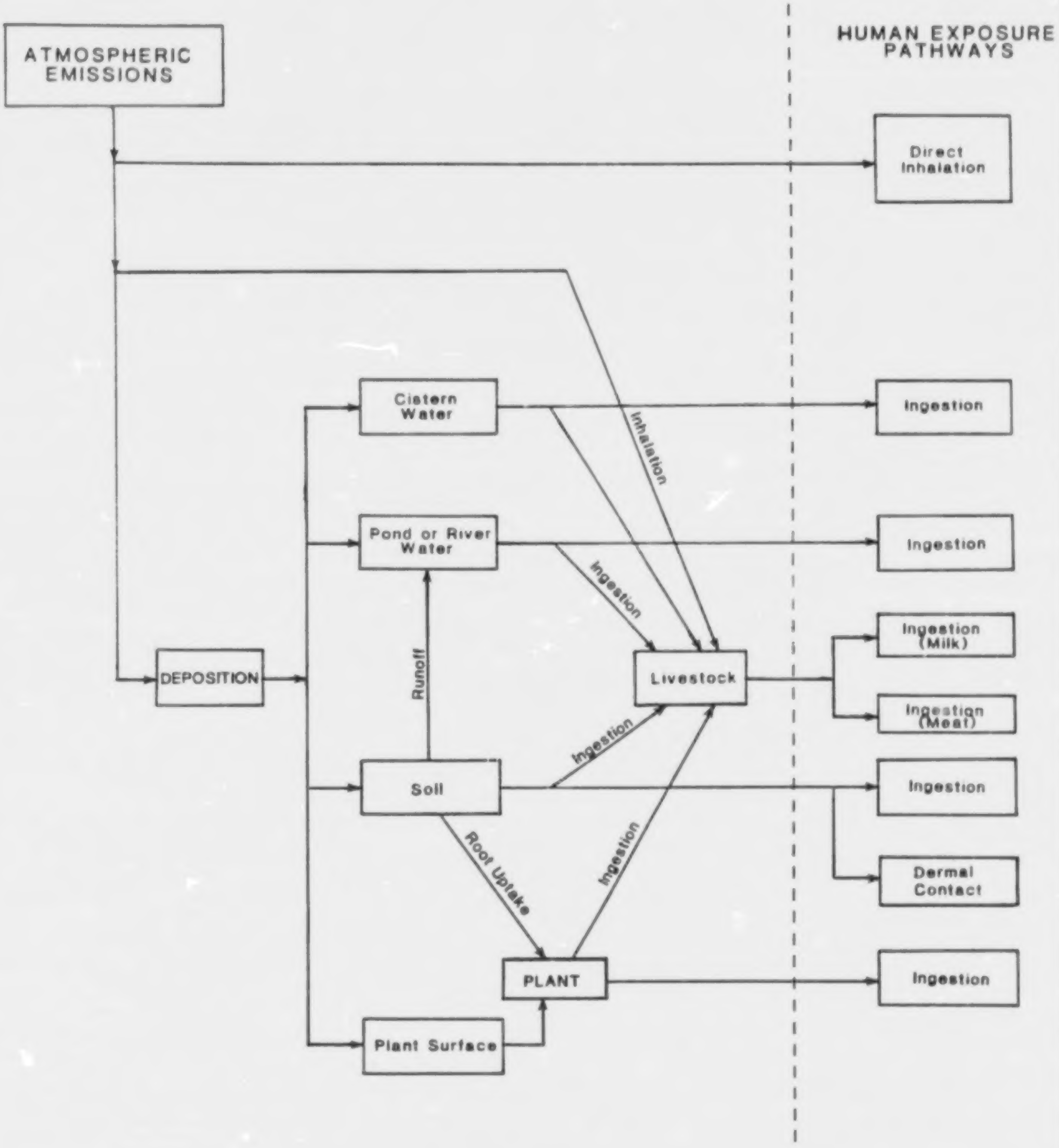
Human exposure to contaminants released to the atmosphere from the proposed OWMC Facility can occur through a variety of both direct and indirect pathways. Direct pathways are defined as those involving exposures to chemicals present in the medium to which the release originally occurs. For example, direct exposure to stack emissions occurs via inhalation of contaminated air; direct exposure to leachate from a landfill occurs via ingestion of contaminated ground water.

Indirect exposures result from the movement of chemicals from one environmental medium to another. For example, contaminants initially released from the incinerator stack to the atmosphere may deposit onto the soil and eventually be taken up by a plant. This plant may then be consumed by an individual, resulting in indirect human exposure to the stack emissions. Alternatively, the plant may be ingested by livestock, possibly resulting in contamination of meat or milk products.

It is not feasible to quantitatively evaluate each human exposure pathway that may be associated with routine atmospheric releases from the proposed OWMC Facility. Exposure pathways were therefore selected for evaluation because they were judged to pose potentially significant risks to human health on the basis of site-specific information (see Chapter 3), or were specifically identified as a public concern. The pathways evaluated in the risk assessment were:

- Direct Inhalation of Air
- Consumption of Crops
    - Leafy vegetables
    - Exposed produce
    - Unexposed produce
- Consumption of Meat and Eggs
    - Beef
    - Pork
    - Poultry
    - Eggs
- Consumption of Milk Products
- Consumption of Drinking Water
    - Ponds
    - Cisterns
- Incidental Ingestion of Soil
- Dermal Contact with Soil

A schematic diagram depicting these pathways is presented in Figure 5.


ATMOSPHERIC EMISSIONS
HUMAN EXPOSURE PATHWAYS
Direct Inhalation
Cistern Water
Ingestion
Inhalation
Pond or River Water
Ingestion
Ingestion
Ingestion (Milk)
Runoff
DEPOSITION
Livestock
Ingestion (Meat)
Ingestion
Soil
Ingestion
Root Uptake
Ingestion
Dermal Contact
PLANT
Ingestion
Plant Surface

HUMAN EXPOSURE
TO ATMOSPHERIC EMISSIONS

Figure
5

5.4 TRANSPORT AND FATE OF CONTAMINANTS RELEASED TO THE ENVIRONMENT

In order to assess the level of risk associated with the human exposure pathways discussed in Section 5.3, it was necessary to estimate the transport and fate of chemicals released to the environment from the proposed OWMC Facility. A general description of the approaches used in estimating the transport and fate of chemicals released to the atmosphere during normal operation is provided in this section. The detailed methodology used in the risk assessment is presented in Appendix D.

The analysis of the environmental transport and fate of contaminants released to the atmosphere during normal operations of the proposed OWMC Facility consisted of the following steps:

- Estimation of atmospheric dispersion and chemical concentrations in air,
- Estimation of atmospheric deposition and chemical concentrations in soil,
- Estimation of chemical concentrations in ponds, rivers, and cisterns, and
- Estimation of chemical concentrations in potential sources of food, e.g., vegetables, milk, eggs, and meat.

These steps are described below.

5.4.1 CHEMICAL CONCENTRATIONS IN AIR

Chemicals released from the proposed facility into the atmosphere will be dispersed as they move from the source of emission. The nature and extent of this dispersion is dependent upon various parameters, including the elevation from which the release occurs and certain meteorological conditions (e.g., wind speed and atmospheric stability).

In the risk assessment, contaminant concentrations in air were estimated by the Ontario Research Foundation (ORF) using the United States Environmental Protection Agency's Industrial Source Complex (ISC) model (ORF 1987). The ISC model has been used to calculate contaminant concentrations in air at an array of locations in the vicinity of the preferred site. Meteorological data based, in part, on monitoring information gathered at the site were used as input to the model.

As previously mentioned, the height from which an emission occurs will influence the extent of contaminant dispersion in air. For incinerator emissions, a stack height of 60 meters was assumed; a stack height of 45 meters was assumed for the evaporator. A height of 25 meters was used in evaluating fugitive emissions vented from the roof of the physical/chemical treatment plant; other fugitive releases were assumed at or near ground level. These elevations are based on the proposed design of the facility (Monenco 1987).

Different time averaging period were used in estimating the dispersion of carcinogens and non-carcinogens in the risk assessment. For carcinogens, the lifetime dose is used in calculating risk, and therefore maximum annual average concentrations in air are used to estimate the level of inhalation exposure. For non-carcinogens, however, the estimated daily dose is compared to a "safe" daily dose, known as the Acceptable Daily Intake (ADI). It has been assumed in the risk assessment that a two-week exposure in excess of the Acceptable Daily Intake (ADI) may result in a toxic effect. Maximum two-week average concentrations in air were, therefore, used to estimate the level of inhalation exposure to non-carcinogens. This assumption is conservative since the maximum two-week average concentrations are higher than maximum annual average concentrations. For exposures to chemicals via ingestion or dermal absorption, maximum annual air concentrations were used, since a long-term accumulation of the chemical in soil is implied for this pathway.

The annual-average and two-week maximum concentrations of chemicals emitted from the incinerator, evaporator, and fugitive sources are presented in Appendix D.

5.4.2 CHEMICAL CONCENTRATIONS IN SOIL

Chemicals deposited onto the ground may accumulate in surface soils over time. Therefore, the calculation of chemical concentrations in soil must be based on both the deposition rate and the period over which deposition is expected to occur.

In the risk assessment, the rates of chemical deposition resulting from both wet and dry processes were considered. Dry deposition described the contact of particulates and gases in the atmosphere with an available surface through atmospheric turbulence and molecular motion. Wet deposition involves the washout of contaminant from the atmosphere by precipitation. Total deposition of chemicals emitted to the atmosphere from the proposed facility was obtained from the sum of the wet and dry deposition processes. Soil concentrations were calculated assuming that deposition occurs for a 70-year period, which was assumed to represent the lifetime of the proposed facility.

Various processes may contribute to the removal of a chemical from the surface soils. These processes include volatilization, leaching to greater depths, soil loss caused by precipitation, uptake by plants, reentrainment to the atmosphere, and biological and chemical degradation. In the Phase 4B risk assessment, it was possible to quantitatively evaluate the following removal mechanisms:

- Volatilization,
- Leaching, and
- Soil loss.

A consideration of these removal mechanisms has been incorporated into the calculation of chemical concentrations in surface soils. Other potential processes through which chemicals may be removed from surface soils were not evaluated due to limitations in available data. The chemical concentrations in soil resulting from a 70-year accumulation of contaminants emitted from the incinerator, evaporator, and fugitive sources are presented in Appendix D.

5.4.3 CHEMICAL CONCENTRATIONS IN CISTERNS

A cistern is a closed reservoir, usually underground, for the collection of rainwater which falls onto rooftops. As noted in Chapter 3, many residences in the vicinity of the proposed facility use cisterns as a source of drinking water. An analysis was therefore performed to estimate the concentrations of the chemicals of concern in cistern water.

Chemical concentrations in cistern water were assumed to be controlled by one of two different processes, depending on whether gaseous or particulate emissions were under examination. For gaseous pollutants, the partitioning between water and air was estimated. For substances which are expected to be absorbed to particulates, concentrations were estimated based on the mass of chemical deposited into the cistern and subsequently released to the water by desorption. As a conservative assumption, it was assumed that the ratio of the mass of a chemical in solution to the mass adsorbed onto particulate would be one.

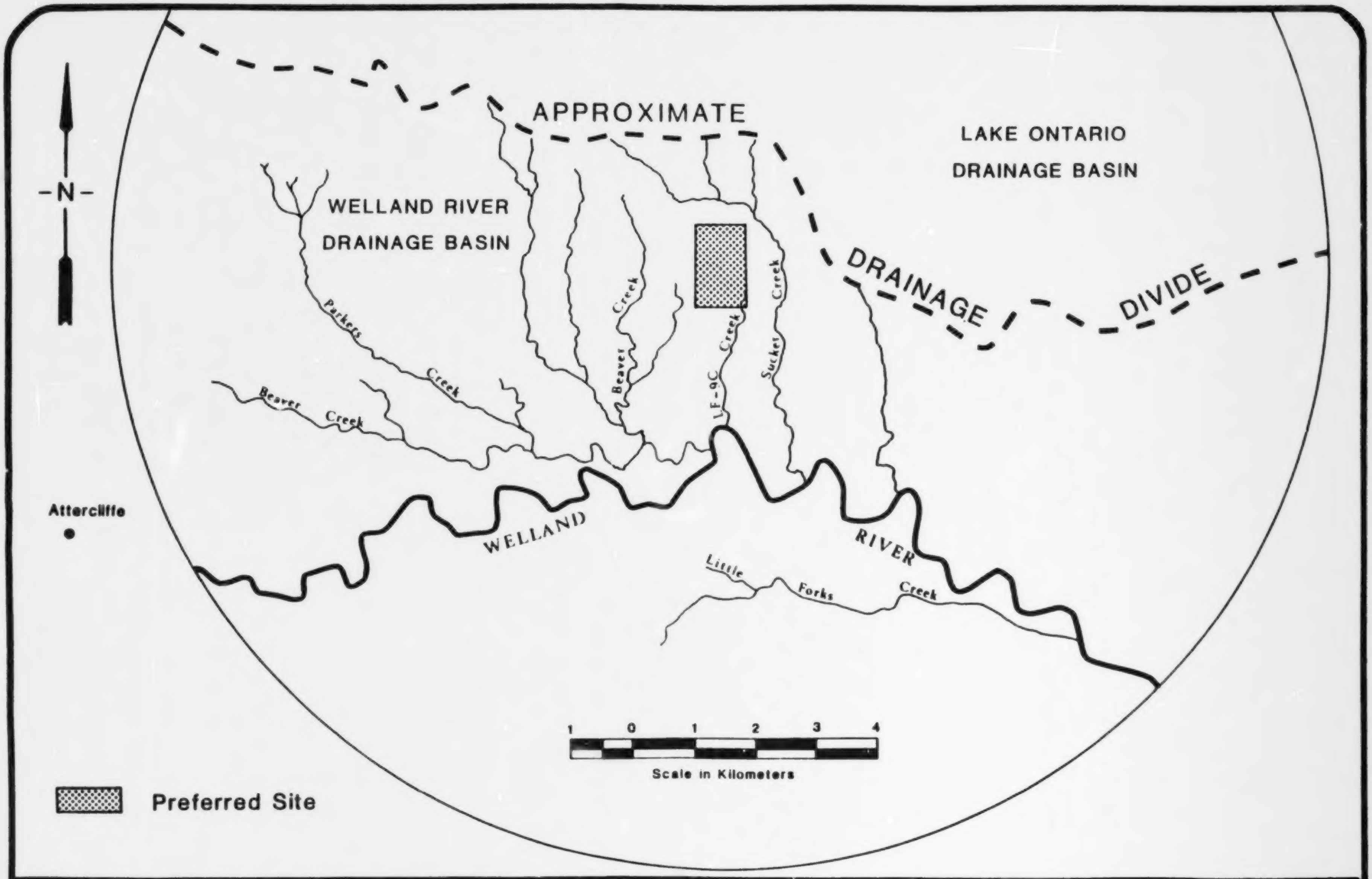
The chemical concentrations in cistern water predicted for incinerator, evaporator, and fugitive emissions are shown in Appendix D.

5.4.4 CONCENTRATIONS IN THE WELLAND RIVER

Chemicals emitted from the OWMC Facility can reach the Welland River primarily through two pathways: (1) direct atmospheric deposition onto the water, and (2) the influx of contaminated runoff into the river.

Contaminated runoff into the Welland River is expected to occur from the portion of the drainage basin shown in Figure 6. Since the drainage basin for the Welland River covers a significantly larger area than the river itself, only contributions from runoff have been considered in the risk assessment.

An annually-averaged, areally-averaged approach was used in evaluating chemical concentrations in the Welland River. It is recognized that the use of averaged parameters in the model may under-predict or over-predict the concentration in the


-N-
APPROXIMATE
DRAINAGE
DIVIDE
LAKE ONTARIO
DRAINAGE BASIN
WELLAND RIVER
DRAINAGE BASIN
Parkers Creek
Beaver Creek
Beaver Creek
LF-9C Creek
Sucker Creek
WELLAND
RIVER
Little Forks Creek
Attercliffe
1 0 1 2 3 4
Scale in Kilometers
Preferred Site

ENVIRON

Counsel in Health and Environmental Science

Drainage Basin for the Welland River

Based on M. M. Dillon, 1987

Figure 6

river at any one time. For example, variations in contaminant concentrations in the river may occur during the winter, when little inflow of pollutants to the river is expected; snowmelt events, when high concentrations of pollutants may exist; and summer storms, which may also result in high chemical concentrations in the river water. However, the approach used in the risk assessment is believed to be appropriate in evaluating long-term exposures.

In calculating runoff into the Welland River, it was assumed that all chemicals emitted to the atmosphere would be deposited in a portion of the drainage basin for that river. This assumption is conservative, since some of the releases from the facility would be expected to deposit in the watershed for Lake Ontario and, therefore, would not contribute to chemical loadings to the Welland River. Average concentrations in soil in the drainage basin were then calculated by incorporating the loss terms described previously in Section 5.4.2.

The mechanism for the movement of pollutants from the surface soil to the river was assumed to be through transport of soil particles due to precipitation and subsequent washoff. Annual average chemical concentrations in the Welland River were estimating based on the yearly inflow of contaminated runoff. In calculating the concentrations in the river, existing levels of metals and phenols in the water were added to the amounts predicted to be contributed by the operation of the OWMC Facility. These existing "background" concentrations, which were discussed in Chapter 3, are significantly higher than the levels associated with routine OWMC emissions. The chemical concentrations in the Welland River, considering both the existing "background" levels and the contribution associated with the operation of the OWMC Facility, are presented in Appendix D for incinerator, evaporator, and fugitive emissions.

Acidification of lakes throughout eastern Canada has been widely attributed to the deposition of acid gases, such as sulfur dioxide (SO_2), emitted from power plants, steel mills, and copper smelters. A simple model was therefore developed to predict the impact of acid gas emissions from the OWMC incinerator on the pH of the Welland River. The basis for this model was the same as that for estimating chemical concentrations in the river, except that all emissions were assumed to be washed into the river each year. In addition, the buffering capacity of the river was also considered by assuming that essentially all of the observed alkalinity naturally occurring in the river is carbonate alkalinity available for reacting with the acid gases. This assumption is considered appropriate given the existence of the dolomite bedrock in the region.

5.4.5 CHEMICAL CONCENTRATIONS IN PONDS

As noted in Chapter 3, there are numerous ponds in the vicinity of the proposed site. Chemicals emitted from the OWMC Facility can reach these nearby ponds either through direct atmospheric deposition or through the influx of runoff. In the risk assessment, both mechanisms were evaluated.

For the purposes of the risk assessment, a hypothetical "typical" pond was defined to reflect the characteristics of the ponds located in the vicinity of the proposed facility. It was conservatively assumed that this pond is located at the point of maximum air concentrations for each routine release from the facility to the atmosphere. This assumption is likely to lead to overestimates of the chemical concentrations in the hypothetical pond.

An annually-averaged, steady-state model was used to predict contaminant concentrations in the hypothetical pond. As discussed in Section 5.4.4, this approach may lead to under- or overestimates of the concentrations in the pond at any one time. It was, however, judged to be appropriate for evaluating long-term exposures.

In calculating runoff into the hypothetical pond, the following assumptions were made:

- The pond is used as a source of drinking water by both humans and livestock for nine months of the year;
- Infiltration of ground water into the pond does not occur;
- Water percolates out through the bottom of the pond;
- The transport of chemicals into the pond is through the movement of soil particles in runoff.

Chemical concentrations in the hypothetical pond were based on the annual inflow of contaminated runoff and direct deposition from the atmosphere. These concentrations are presented in Appendix D. Background chemical concentrations in ponds near the proposed site were not available and therefore could not be considered in the risk assessment.

5.4.6 CHEMICAL CONCENTRATIONS IN PLANTS

Human exposure to chemicals emitted from the proposed OWMC Facility may occur through a variety of food pathways. The ingestion of a variety of fruits and vegetables, beef, milk, pork, poultry, and eggs have been considered in the risk assessment in estimating total human exposures.

In order to predict the potential risks associated with the consumption of these foods, the concentrations of chemicals in plants ingested directly by humans were estimated. Furthermore, since certain crops are used as feed for cattle, swine, and poultry, contaminant concentrations in such produce were also calculated. (See Section 5.4.7 for a discussion of the relevance of chemical concentrations in plants in estimating contaminant levels in meat, milk, and eggs.)

In the risk assessment, chemical concentrations in the following types of produce have been examined:

- Leafy vegetables, which present a leafy, edible surface exposed to the atmosphere. Examples include lettuce and broccoli.
- Exposed produce, which also present edible surfaces exposed to the atmosphere, but which are not leafy in nature. Examples include tomatoes and apples.
- Unexposed produce, which do not have edible surfaces exposed to the atmosphere. Examples include potatoes and corn.

Livestock have been assumed to consume hay, mixed grains, and/or corn silage in the risk assessment.

Chemicals may accumulate in a plant as the result of several mechanisms, including direct deposition onto exposed surfaces and uptake from the soil through the root system. Both of these mechanisms have been quantitatively evaluated in the risk assessment. A third pathway, foliar absorption of vapor-phase organics, was also considered.

The extent to which deposition plays a significant role is dependent on the characteristics of both the chemical and the plant considered. Direct deposition onto a plant surface would not contribute to the accumulation of chemicals in the edible portion of unexposed produce unless translocation is significant. The rate of chemical uptake from soil is also a function of the type of plant and the nature of the contaminant. For example, chemicals which are highly soluble in water are predicted to be taken up more readily by the root system than those which are relatively insoluble. In addition, the vegetative parts (e.g., leaves, stems, and straws) of plants tend to accumulate chemicals to a greater degree than the reproductive portions (e.g., fruits, seeds, and tubers) (Baes et al. 1984).

Several processes may reduce the degree to which atmospheric deposition contributes to the accumulation of chemicals in a plant. These processes include photolysis (the breakdown of a chemical due to exposure to sunlight), volatilization, and washoff resulting from precipitation. Of these mechanisms, photolytic degradation could not be quantitatively evaluated in the risk assessment due to limitations in available data. The impacts of losses due to volatilization and washoff, however, were examined by calculating the chemical concentrations in plants resulting from direct deposition onto plant surfaces. Chemical concentrations in the various plants evaluated in the risk assessment are shown in Appendix D.

5.4.7 CHEMICAL CONCENTRATIONS IN MEAT, MILK, AND EGGS

As discussed in Section 5.3, the ingestion of food is a potentially significant pathway for human exposure to chemicals in the environment. To estimate the extent to which this pathway may lead to exposure to emissions from the proposed OWMC Facility, ingestion of beef, pork, poultry, milk, and eggs was evaluated in the risk assessment.

To determine contaminant concentrations in milk, meat, and eggs, it was first necessary to calculate exposures to cattle, swine and poultry. In calculating these exposures, the following pathways were examined:

- Consumption of crops,
- Ingestion of river water, and
- Inhalation of air.

For cattle, ingestion of soil was also evaluated. Based on consideration of these exposure pathways, a total systemic dose for each of the livestock of concern was estimated.

From the estimated systemic dose, it was possible to calculate the fraction of each chemical retained in the various animal products, i.e., beef, milk, pork, poultry, and eggs. This was accomplished through the use of biotransfer factors to relate the concentration in the animal product to the estimated dose. These biotransfer factors vary widely according to the nature of the chemical considered. For example, certain environmentally persistent, relatively water-insoluble chemicals, such as polychlorinated biphenyls (PCBs), accumulate in animal tissues and milk to a much greater extent than other contaminants.

For most contaminants, it was necessary to use generalized correlations based on physical/chemical properties to predict the biotransfer of chemicals to the animal products examined in the risk assessment. Experimentally observed values, however, have been adopted for those chemicals, e.g., PCBs and hexachloro- benzene, for which data was available. In addition, a detailed review of data relating to accumulation of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in beef and milk was conducted (see Appendix F). This review was undertaken in response to the findings of the Phase 4A Generic Risk Assessment, which indicated that the ingestion of milk and beef contaminated with PCDD/PCDF has the potential to contribute significantly to the risks associated with the operation of the proposed OWMC Facility.

Chemical concentrations in the various animal products considered in the risk assessment are presented in Appendix D. It should be noted that, in general, the partitioning of chemicals to beef and milk has been more closely studied than the potential for accumulation in pork, poultry and eggs. For this reason, greater uncertainty is likely to be associated with the estimation of chemical concentrations in those animal products other than milk or beef. It should also be recognized that the feed for some farm animals, such as poultry, may often be imported from areas not likely to be impacted by facility emissions.

5.5 ESTIMATION OF HUMAN EXPOSURES

Once contaminant concentrations in the various environmental media have been calculated, it is possible to estimate human exposures for each pathway of concern. This step of the risk assessment process typically involves the application of conservative assumptions to allow for an estimation of the total systemic dose for potentially exposed individuals.

The level of exposure to contaminants released from the OWMC Facility is dependent upon a number of factors, including an individual's location, diet, and behavior. For example, contaminants emitted from the incinerator stack will be dispersed in the atmosphere; the highest annually-averaged chemical concentrations in air associated with these emissions are predicted to be located at a distance of approximately

1 kilometer northeast of the facility. A person located at this point is likely to inhale a greater quantity of a given contaminant released from the incinerator than someone situated elsewhere in the site vicinity. Furthermore, higher contaminant doses are predicted the longer one is exposed to the stack emissions. Clearly, persons located at different locations in the site vicinity for different periods of time would not be expected to receive the same dose of a chemical emitted from the incinerator stack.

However, it is usually impossible to accurately estimate the likely level of exposure for each individual in a large community because of variations and uncertainties in both current and future behavior patterns. Therefore, conservative assumptions, which are likely to overestimate the level of human exposure, are often made in conducting a risk assessment.

In the current analysis, a number of conservative assumptions have been made in predicting both long-term and human exposures. These assumptions are summarized below:

- For exposures via inhalation, all dose estimates are based on a person located at the point of highest off-site ground-level contaminant concentrations.
- For exposures resulting from ingestion of (or dermal contact with) soil, all dose estimates are based on a person permanently located at the point of highest off-site ground-level contaminant concentrations.
- 100 percent of the soil ingestion and contact is assumed to occur at the location of maximum off-site contamination.

- For exposures resulting from ingestion of crops, all dose estimates are based on the consumption of produce grown at the location of highest off-site ground-level contaminant concentrations.
- 100 percent of the produce consumed by a person is assumed to be taken from the location of maximum off-site contamination.
- For exposures resulting from ingestion of meat, all dose estimates are based on the consumption of livestock and poultry raised at the point of highest off-site contaminant concentrations. Fodder for these animals is assumed to be taken from the same point; water is assumed to be taken from the Welland River.
- 100 percent of the beef, pork, poultry, and eggs consumed by a person is assumed to be taken from the location of maximum off-site contamination.
- For exposures resulting from the ingestion of surface water, all dose estimates are based on the consumption of water taken from a pond or cistern located at the point of highest off-site ground-level contaminant concentrations.
- 100 percent of the water ingested by the person is assumed to be taken from a pond or cistern located at the point of maximum off-site contamination.
- All long-term exposures are assumed to occur continuously for a period of 70 years.

A number of assumptions regarding the diet of potentially exposed individuals was also necessary. These assumptions were based primarily on data collected by Statistics Canada (1986 and 1987) and are summarized in Table 5-1.

As shown in Table 5-1, body weights and breathing rates are different for children than they are for adults. Similarly, the amount of food consumed by a child would not be the same as for an adult. For this reason, the chemical dose received by a

TABLE 5-1

STANDARD ASSUMPTIONS FOR HUMAN DOSE CALCULATIONS[1]

Body weight (adult)	70	kg	NIOSH 1980
Body weight (child)	31	kg	ICRP 1984
Breathing rate (adult)[2]	23	m^3/d	Altman and Dittmer 1972
Breathing rate (child)[3]	12	m^3/d	ICRP 1984
Water consumption	2.0	l/d	USEPA 1980
Beef consumption	0.106	kg/d	Statistics Canada 1986
Pork consumption	0.178	kg/d	Statistics Canada 1986
Poultry consumption	0.069	kg/d	Statistics Canada 1986
Egg consumption	0.033	kg/d	Statistics Canada 1986
Milk consumption	0.381	kg/d	Statistics Canada 1986
Exposed produce consumption	0.178	kg/d	Derived from Statistics Canada 1987
Protected produce consumption	0.271	kg/d	Derived from Statistics Canada 1987
Leafy vegetable consumption	0.033	kg/d	Derived from Statistics Canada 1987
Incidental soil ingestion (child)	100	mg/d	USEPA 1984
Incidental soil ingestion (adult)	20	mg/d	USEPA 1984

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[1] All values correspond to an average adult unless otherwise noted.

[2] Value used represents a man doing light work for ten hours (1.7 m^3/h) and at rest for 14 hours (0.4 m^3/n)

[3] Value used represents a child performing light activity for ten hours (0.78 m^3/h) and at rest for 14 hours (0.28 m^3/h)

child may differ from the dose received by an adult. In most cases, a young child will receive a somewhat higher dose per kilogram body weight than will an adult. For 70-year chronic exposures to carcinogens, however, the fraction of time an individual is a child is small relative to the total period of exposure. Chronic exposures to carcinogens were thus generally evaluated by examining the dose predicted to be received by an adult. The dose corresponding to dermal contact with, and ingestion of, soil were calculated separately for children and adults, however, since children may exhibit significantly different patterns of exposure through these pathways.

In evaluating non-carcinogenic impacts, it has been conservatively assumed in the risk assessment that an exposure period as short as two weeks could result in the manifestation of adverse health effects. Such a two-week exposure could occur to either a child or an adult. For this reason, exposures to non-carcinogens were considered for children as well as for adults, although the base case involved an examination of the dose received by an adult.

As discussed in Chapter 2, non-carcinogens are typically assumed to act via a threshold mechanism; i.e., that a certain dose must be exceeded before adverse human health effects would be anticipated. In some cases, emissions from the OWMC facility will add to an existing level of exposure that is associated with "background" contamination in air, soil, and other environmental media. For this reason, two analyses have been performed in evaluating the risks associated with exposures to non-carcinogens:

- In the base case, only the dose resulting from the operation of the OWMC facility was examined. (Existing levels of contaminants in the Welland River were also considered, but these concentrations do not have a significant impact on the human health risk assessment.)
- In a separate evaluation, available data with respect to existing chemical concentrations in air and soil were considered in developing an estimate of the total dose associated with exposures to both facility emissions and "background" contamination. These data are summarized in Chapter 3.

The location of the maximum ground level contaminant concentrations is different for incinerator, evaporator, and fugitive releases. Therefore, exposures corresponding to each of these three types of emissions have been calculated separately. Calculations for chemical exposures associated with each source of release and pathway examined are presented in Appendix I along with the estimated levels of exposure for the base case analysis.

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CHAPTER 6

THE RELEASE OF LEACHATE FROM THE ENGINEERED LANDFILL

6.1 INTRODUCTION

The proposed OWMC Facility will include an engineered landfill for the disposal of solid residues produced by various on-site treatment processes (Monenco 1987b). These residues will consist of:

- Approximately 2 700 t/a of slag (a fused material composed primarily of metal compounds) generated in the rotary kiln incinerator;
- Approximately 12 000 t/a of solidified material from an incinerator solidification unit. This solidification unit will receive approximately 6 500 t/a salt and ash from the waste heat boiler, spray dryer, and electrostatic precipitator components of the incineration system. The salt and ash will be mixed with various reagents (e.g., Portland cement and lime) to produce a solid material.
- Approximately 99 000 t/a of stabilized wastes from the main solidification plant. The main solidification plant will receive solid inorganic wastes from off-site generators and solid residues generated in the physical/chemical treatment plant. These wastes will be mixed with various reagents (e.g., Portland cement, sodium silicate solution, and lime) to produce a solid material.

Each of these three types of waste will be composed primarily of inorganic constituents, such as silica and metals, although small amounts of organic chemicals may also be present.

The purpose of the landfill is to contain potentially hazardous chemicals that may be present in the incinerator residues and solidified wastes. However, in practice the compacted clay cover placed on the landfill will allow the infiltration of small quantities of rainfall or snowmelt. As this water percolates through the landfill, chemicals will dissolve out of the incinerator residues and the solidified waste to generate an aqueous liquid known as leachate. Subsequent migration of leachate from the landfill, either laterally to surface water or downward to ground water, may result in contamination of the off-site environment.

In this chapter, the general approach used to evaluate the potential human health risks associated with the release of leachate from the engineered landfill is discussed. The procedures used to estimate the concentrations of chemicals in the leachate from the OWMC landfill are described in greater detail in Appendix C.

6.2 CONTAMINANT RELEASES FROM THE OWMC LANDFILL

6.2.1 INTRODUCTION

The first step in assessing the potential public health risks associated with releases from the OWMC landfill was to estimate of the contaminant concentrations in the leachate. This process consisted of two steps:

- Characterization of the chemical composition of the wastes to be deposited in the OWMC landfill; and
- Estimation of the release of specific chemicals from the landfilled wastes to the leachate.

These two steps are discussed in the following subsections of this chapter.

6.2.2 CHARACTERIZATION OF WASTES

As discussed in Section 6.1, the wastes to be placed in the landfill will consist of (1) incinerator slag; (2) solidified incinerator salt and ash; and (3) wastes produced by the OWMC solidification plant. Since the chemical composition of these three types of residues will be different, they were characterized separately in the landfill analysis.

6.2.2.1 Incinerator Slag

Information on the constituents present in slag produced by existing hazardous waste incinerators has been compiled by the engineering consultants to OWMC (Monenco 1986). These data were generated at three incinerators (Kommunekemi in Denmark, HIM Biebesheim in Germany, and SAKAB in Sweden) which are similar in design and operation to the proposed OWMC Facility. These data were considered to represent the "typical" composition of slag for the purposes of the risk assessment. It should be recognized, however, that the chemical composition of slag may vary significantly depending on the characteristics of the waste being incinerated. To allow for this type of uncertainty, the highest concentration reported for each contaminant at the three facilities was used in the assessment.

The inorganics reported in the incinerator slag from the Kommunekemi, HIM, and SAKAB facilities were categorized into three groups:

- Potentially hazardous inorganics for which available toxicity data are sufficient for the purposes of risk assessment. These contaminants include cadmium, lead, and chromium.
- Potentially hazardous inorganics for which available toxicity data are insufficient for the purposes of risk assessment. These contaminants include aluminum, manganese, cobalt, and boron.
- Inorganics, such as iron, sodium, and potassium, that pose relatively low public health hazards.

To allow for the absence of adequate toxicity data for a portion of the potentially hazardous material, inorganics for which sufficient toxicity data are available were selected as surrogates. The quantities of those substances which may be hazardous, but for which adequate toxicity data are not available, were represented by these surrogates in the risk assessment. Concentrations and quantities of the surrogate inorganic chemicals in the slag are presented in Table 6-1.

6.2.2.2 Incinerator Salt and Ash

Data on the potentially hazardous constituents of incinerator salt and ash have also been reported by the engineering consultants to OWMC (Monenco 1986). These data are from only one facility (HIM Biebesheim in West Germany) and are limited to inorganic constituents. In using these data, the approach described in Section 6.2.2.1 to allow for the absence of adequate toxicity data on a portion of the potentially hazardous material was also applied to characterize the inorganic content of salt and ash. The resulting concentrations of inorganic contaminants in salt and ash are summarized in Table 6-2.

Data provided by the engineering consultants to OWMC indicates that it is likely that a small portion (less than 1 percent) of the salt and ash produced by the incinerator will

TABLE 6-1

CONCENTRATIONS OF POTENTIALLY HAZARDOUS CHEMICALS IN INCINERATOR SLAG

Contaminant	Concentration[1] (ppm)	Quantity[2] (t/a)
Barium	103 090	278
Copper	81 390	220
Chromium	106 890	289
Nickel	5 430	15
Zinc	2 713	7.3
Lead	5 430	15
Silver	30	0.07

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[1] Derived from data reported in Monenco (1986, 1987b).

[2] Based on 2 700 t/a slag produced by rotary kiln incineration (Monenco 1986, 1987b).

See Appendix C for a detailed description of the methodology used to develop the quantity estimates presented in this table.

TABLE 6-2

CONTAMINANT CONCENTRATIONS IN INCINERATOR SALT AND ASH

Contaminant	Concentration[1] (ppm)	Quantity (t/a)[3]
Cadmium	330	2.2
Mercury	20	0.1
Barium	380	2.5
Copper	1 080	7.0
Chromium	210	1.3
Nickel	170	1.1
Zinc	11 940	77.6
Lead	6 480	42.1
Nitrate	40	0.26
PCDD/PCDF[2]	0.041	2.7×10^{-4}

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[1] Data for inorganic contaminants derived from Monenco (1986, 1987b); estimate of PCDD/PCDF concentration developed by ENVIRON (see Appendix C).

[2] Polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) expressed as 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents (TEQ). See Appendix H for a discussion of TEQ factors.

[3] Based on 6 500 t/a salt and ash produced by the rotary kiln incinerator (Monenco 1987b).

See Appendix C for a detailed description of the methodology used to develop the quantity estimates presented in this table.

consist of organic material (Monenco 1986). These data, however, do not identify specific organic chemicals that may be expected. It is known, however, that salt and ash collected in the boiler and air pollution equipment may contain hazardous organics that condense onto the particulates in the post-combustion zone of the incinerator.

Of the organics that may be present in incinerator salt and ash, public health concerns have focused primarily on a family of compounds known as polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) since they are generally believed to be very potent carcinogens and are known to be produced during some combustion processes (see, for example, CARB 1984). There are, however, very few data on PCDD/PCDF concentrations in salt and ash that have been collected at incinerators similar in design and operation to the OWMC Facility, i.e., HIM Biebesheim, Kommunekemi, and SAKAB. Given this limitation, an alternative approach to the use of data from existing facilities was developed to provide an estimate of PCDD/PCDF concentrations in salt and ash from the OWMC incinerator. In this approach, it was assumed that the particulate matter removed in the air pollution control system would have the same PCDD/PCDF concentration as the particles emitted from the incinerator stack. This concentration was calculated by dividing the predicted PCDD/PCDF emission rate (expressed as 2,3,7,8-TCDD equivalents [see Appendix H] in mg per normal cubic meter of flue gas) by the expected particulate emission rate (in mg particulate per normal cubic meter of flue gas). Resulting concentrations of PCDD/PCDF in salt and flyash are shown in Table 6-2.

6.2.2.3 Residues Generated by the Solidification Plant

Residues produced by the OWMC solidification plant will represent the largest quantity of material deposited in the engineered landfill. The solidification plant will mix solid inorganic wastes received from off-site generators, solid residues generated by the physical/chemical treatment plant, and various reagents to produce a physically stable matrix designed to limit infiltration and leachate generation.

In order to characterize the composition of the residues produced by the solidification plant, it was first necessary to determine the identity and concentration of chemicals in both the wastes to be received from off-site generators and in the solid residues produced by the physical/chemical plant. The methodology developed for estimating contaminant concentrations for these two sources of waste are outlined below.

Chemical Loadings from Directly Solidified Off-Site Wastes. The estimation of contaminant concentrations in the wastes to be sent directly to solidification without treatment in the physical/chemical plant is described in Chapter 4 of this report. The data used to estimate contaminant concentrations in the wastes were derived from studies conducted by CANVIRO Consultants (1985) and Franklin Associates. These contaminant concentrations were multiplied by the total quantity of waste to develop estimates of chemical loadings to the solidification unit from off-site generators. The quantities of surrogate chemicals in these wastes are shown in Table 6-3.

Chemical Loadings from Wastes Initially Treated in the Physical/Chemical Plant. To estimate the quantities of specific chemicals in the solid residues sent from the physical/chemical treatment plant to the solidification unit, a more complex procedure was required. This procedure involved: (1) prediction of the identity and quantity of specific chemicals in the inorganic wastes to be received by the physical/chemical treatment plant, and (2) an analysis of the performance of the various physical/chemical processes in treating these wastes.

In the analysis, it was assumed that virtually all inorganic contaminants in the wastes to be treated in the physical/chemical plant would ultimately reside in the solid residues sent to the solidification unit. An exception would be cyanide, which will be effectively destroyed in the chemical oxidation unit. The quantities of most inorganic chemicals in the residues sent to solidification were therefore assumed to

TABLE 6-3

SURROGATE CHEMICALS IN THE INORGANIC WASTES SENT DIRECTLY TO THE SOLIDIFICATION UNIT

Chemicals	Quantity (t/a)
Metals	
Arsenic	0.08
Barium	0.05
Cadmium	4.1
Chromium	304.4
Copper	912.9
Lead	923.4
Mercury	2.6
Nickel	229.0
Silver	0.98
Zinc	1765.4
Inorganics	
Fluoride	0.01
Nitrate	4.9
Organics	
Benzo(a)anthracene	0.006
Benzo(a)pyrene	0.003
Benzene	0.04
Cresols	0.15
Dichlorodifluoromethane	0.1
Ethanol	0.1
Formaldehyde*	3.1
Naphthalene	0.12
PCBs	0.007
Petroleum distillates	3.9
Phenols*	5.54
Tetrachloroethylene	0.25
Toluene	58.3
1,1,1-Trichloroethane	0.53
Trichloroethylene	0.12
Trichlorotrifluorethane	0.19
Xylene	0.75

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* According to the engineering consultants to OWMC, approximately 95 percent of the phenol and formaldehyde reported in the CANVIRO data base is in the form of a phenol-formaldehyde resin (Monenco 1987a). Since such a resin would have much different leaching characteristics than phenol or formaldehyde, the engineering consultants have adjusted the estimated loadings to represent only the portion of these chemicals that may be liquid.

be the same as the quantities received by the physical/chemical treatment plant. The approach used to develop the amounts of specific inorganic constituents in the wastes to be treated at the physical/chemical plant is discussed in Chapter 4 of this report.

For the organic contaminants in the waste feed to the physical/chemical treatment facility, the extent to which an organic will partition to the solid residues is dependent, in part, upon its physical and chemical properties. Non-volatile, high molecular weight organic chemicals, such as PCBs, would be expected to adsorb to the solid residues and therefore would likely be sent to the solidification plant. Other organic constituents, however, will be separated from the solids during certain physical chemical processes. For example, volatile organics such as acrylonitrile will be largely removed in the emulsion breaking and air stripping units, while low molecular weight, highly soluble chemicals such as ethanol will tend to remain in solution.

In order to predict the concentrations of specific organics in the solid residues generated by the physical/chemical plant, the treatment performance for organic contaminant separation was assessed on an individual stream basis. This assessment was performed by the engineering consultants to OWMC (Monenco 1987a) using treatability data available in the literature or generated by tests commissioned by OWMC (Zenon 1987). These tests were undertaken to evaluate the performance of the various physical/chemical plant process units in treating different types of organic substances. Information provided in the CANVIRO data base was used to identify which waste streams to be received at the physical/chemical plant are likely to contain organic constituents, and what the concentrations of the specific chemicals in these streams may be.

The only organic chemicals that were not examined on an individual stream basis were those identified in the Franklin data base as possible constituents of waste oil. Since "oil and grease" is expected in a relatively large number of inorganic streams to be received at the physical/chemical plant, it was not feasible to conduct a stream-by-stream evaluation of the partitioning of organic chemicals in oil to the solid residues. The quantities of oil contaminants likely to reside in the solid residues sent from the physical/chemical treatment plant to the solidification plant were therefore estimated by the engineering consultants to OWMC (Monenco 1987a).

The estimated loadings of chemicals in the residues sent to the solidification plant are presented in Table 6-4. It should be recognized that there is considerable uncertainty associated with these estimates because of limitations in the

TABLE 6-4

SURROGATE CHEMICALS IN THE PHYSICAL/CHEMICAL TREATMENT PLANT RESIDUES SENT TO THE SOLIDIFICATION UNIT

Chemicals	Quantity (t/a)
Metals	
Antimony	6.7
Arsenic	6.8
Barium	0.02
Cadmium	15.5
Chromium	193.5
Copper	153.7
Lead	12.5
Mercury	0.77
Nickel	145.3
Silver	29.6
Zinc	753.4
Inorganics	
Cyanide	0.0005
Fluoride	0.07
Nitrate	1.6
Organics	
Benzo(a)anthracene	0.010
Benzo(a)pyrene	0.004
Benzene	0.076
Dichlorodifluoromethane	0.16
Naphthalene	0.20
PCBs	0.013
Phenols	0.11
Styrene	0.061
Tetrachloroethylene	0.41
Toluene	0.17
1,1,1-Trichloroethane	0.17
Trichloroethylene	0.20
Trichlorotrifluorethane	0.33
Xylene	0.16

See Appendix C for a detailed description of the methodologies used to develop the quantity estimates presented in this table.

data on organic contaminants in the inorganic wastes to be received by the physical/chemical treatment plant.

6.2.1.4 Total Loadings to the Engineered Landfill

The total loadings of specific chemicals to the proposed OWMC engineering landfill were calculated by combining the estimates developed for incinerator slag (Table 6-1), incinerator salt and ash (Table 6-2), and residues produced by the solidification plant (Tables 6-3 and 6-4). These combined loadings are presented in Table 6-5. The methods used to predict the likely concentration of each of these chemicals in the leachate generated in the landfill are discussed in the next section of this chapter.

6.2.3 ESTIMATION OF CHEMICAL CONCENTRATIONS IN LEACHATE

As previously discussed in Section 6.1, the infiltration of precipitation into the OWMC engineered landfill will result in the partial solubilization of contaminants to produce an aqueous liquid known as leachate. As part of the risk assessment, an approach was developed to estimate the concentrations of chemicals in leachate based on the predicted concentrations in the landfilled wastes (WTC 1987). The approach developed in the assessment was based on the following assumptions:

TABLE 6-5
SURROGATE CHEMICALS IN THE SOLIDIFIED WASTES SENT TO THE ENGINEERED LANDFILL

Chemicals	Quantity (t/a)	Mass Fraction in Solidified Waste*
Metals		
Antimony	6.7	5.9 E-05
Arsenic	6.9	6.1 E-05
Barium	280.6	2.5 E-03
Cadmium	21.8	1.9 E-04
Chromium	788.2	7.0 E-03
Copper	1 293.6	1.1 E-02
Lead	993.0	8.8 E-03
Mercury	3.5	3.1 E-05
Nickel	390.1	3.5 E-03
Silver	30.7	2.7 E-04
Zinc	2 603.7	2.3 E-02
Inorganics		
Cyanide	0.0005	4.4 E-09
Fluoride	0.1	7.1 E-07
Nitrate	6.8	6.0 E-05
Organics		
Benzo(a)anthracene	0.02	1.4 E-07
Benzo(a)pyrene	0.01	6.3 E-08
Benzene	0.12	1.0 E-06
Cresols	0.15	1.3 E-06
Dichlorodifluoromethane	0.26	2.3 E-06
Ethanol	0.10	8.8 E-07
Formaldehyde	3.1	2.8 E-05
Naphthalene	0.32	2.8 E-06
PCBs	0.02	1.8 E-07
PCDD/PCDF (2,3,7,8-TEQ)	0.00027	2.4 E-09
Petroleum distillates	3.90	3.5 E-05
Phenols	5.65	5.0 E-05
Styrene	0.06	5.4 E-07
Tetrachloroethylene	0.66	5.8 E-06
Toluene	58.5	5.2 E-04
1,1,1-Trichloroethane	0.69	6.2 E-06
Trichloroethylene	0.32	2.8 E-06
Trichlorotrifluorethane	0.52	4.6 E-06
Xylene	0.91	8.1 E-06

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* Based on 113 000 t/a solidified waste.

- The solidified waste is a porous solid.
- The composition of the pore solution within the solidified waste is equivalent to the composition of the leachate generated in the landfill.
- The pore solution is in continuous chemical equilibrium with the solid portion of the solidified waste.

Since the leachate composition will depend upon the physical and chemical characteristics of the contaminants present in the solidified wastes and residues, chemicals were categorized into three groups:

- Class A. Chemicals which are completely soluble in the pore solution of the solidified material.
- Class B. Chemicals for which the concentration in the pore solution is controlled by a linear adsorption isotherm; i.e., the concentration in the solidified material divided by the concentration in the pore solution is equal to a constant greater than zero. This constant is referred to as the adsorption coefficient (Kd).
- Class C. Chemicals for which the concentration in the pore solution is controlled by the solubility of a slightly soluble salt.

For contaminants in Class A, the chemical concentrations in leachate can be calculated from 1) the mass fraction of the contaminant in the treated residues from the solidification plant, 2) the mass of solidified waste, and 3) the water content of the solidified waste. The formula used to calculate chemical concentrations in leachate using this information is presented in Appendix C.

For contaminants in Class B, an estimate of the adsorption coefficient (Kd) is also required. Since only limited Kd data are available, OWMC commissioned adsorption tests for a variety of organic substances as part of the Phase 4B activities (WTC 1987). Based on the results of these tests, WTC has estimated appropriate Kd values where possible for the chemicals examined in the landfill analysis. These values are presented in Appendix C.

For contaminants in Class C, the pore solution concentration is independent of the concentration in the solidified waste. For these contaminants, the pore solution concentration is determined by the chemical speciation (e.g., carbonate, hydroxide, sulphide (WTC 1987). Equilibrium leaching tests were conducted for certain Class C chemicals considered in the landfill analysis to estimate pore solution concentrations for these contaminants. The results of these leaching tests are presented in Appendix C.

Based on the approach outlined in this section, chemical concentrations in the landfill leachate were estimated. These concentrations are presented in Table 6-6.

TABLE 6-6

SURROGATE CHEMICAL CONCENTRATIONS IN LEACHATE

Chemicals	Leachate Concentration (mg/l)
Metals	
Antimony	0.05
Arsenic	0.01
Barium	0.3
Cadmium	0.01
Chromium	0.01
Copper	0.1
Lead	0.01
Mercury	1.0 E-04
Nickel	0.1
Zinc	0.1
Inorganics	
Cyanide	3.6 E-04
Fluoride	1.8
Nitrate	150
Organics	
Benzo(a)anthracene	1.57 E-04
Benzo(a)pyrene	6.98 E-05
Benzene	0.017
Cresols	0.0044
Dichlorodifluoromethane	5.8
Ethanol	2.2
Formaldehyde*	69
Naphthalene	0.0032
PCBs	5.9 E-05
PCDD/PCDF (2,3,7,8-TEQ)	7.6 E-09
Petroleum distillates	0.12
Phenols	0.83
Styrene	0.009
Tetrachloroethylene	0.097
Toluene	8.6
1,1,1-Trichloroethane	0.20
Trichloroethylene	0.047
Trichlorotrifluorethane	11.5
Xylene	0.013

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* Leachate concentration based on liquid formaldehyde. According to the engineering consultants, actual loadings may be in a polymerized form (Monenco 1987a). Therefore, leachate concentrations may be significantly overestimated.

See Appendix C for a detailed description of the approach used to develop potential leachate concentrations.

6.3 ESTIMATION OF POTENTIAL HUMAN EXPOSURES

In Section 6.2, the approaches used to estimate the concentrations of selected chemicals in the leachate from the proposed OWMC landfill were outlined. In this section, the methodology used to evaluate the potential for human exposure to these chemicals is described using the following steps:

- Identification of those pathways that may contribute significantly to human exposure; and
- Application of fate and transport models to predict chemical concentrations in the relevant environmental media.

The predicted chemical concentrations in the environment can be used to assess human health risk posed by the release of leachate from the OWMC landfill.

6.3.1 EXPOSURE PATHWAYS

To identify the exposure pathways of concern, it was first necessary to consider the potential for leachate release from the landfill. The shallow entombed landfill design selected for the proposed OWMC Facility was identified by the engineering consultants to OWMC as having the least potential for disruption of natural surface and ground water systems (M.M. Dillon 1986). This design minimizes the possibility that leachate generated in the landfill will seep to the surface or enter the shallow ground water flow system. Furthermore, certain site characteristics, such as the 15 to 20 meter clay layer beneath the landfill, minimize the potential for leachate to reach the bedrock aquifer. Travel time through the clay layer has been estimated to be on the order of thousands of years even for those chemicals which do not adsorb strongly to clay; organics which do adsorb strongly (e.g., PCDD/PCDF) would not be expected to reach the aquifer for at least hundreds of thousands of years (Gartner Lee 1987).

Notwithstanding the high degree of natural protection of the bedrock aquifer, it is conceivable that ground water contamination could occur in the distant future. Ingestion of ground water taken from the bedrock aquifer was therefore identified as the human exposure pathway of greatest concern. It should be noted that, although the existing bedrock ground water quality is generally good, there are currently only four bedrock wells in use downgradient between the site and the Welland River (Gartner Lee 1987). Two of these wells are currently used for drinking water purposes.

6.3.2 ENVIRONMENTAL FATE AND TRANSPORT

To estimate the potential for significant ground water contamination, the behavior of chemicals as they travel through the clay layer toward the aquifer beneath the site must be evaluated. If contaminant concentrations in the leachate are not diminished during transport, levels in the ground water could eventually approach those in the leachate. In practice, however, it is expected that several processes, known as attenuation mechanisms, are likely to lead to a significant reduction in leachate concentrations over time (Roy and Griffin 1987). These mechanisms include:

- Dilution. Leachate generated in a landfill will migrate from the site and may eventually mix with ground water. This mixing process, known as dilution, may or may not represent a significant attenuation mechanism, depending on the quantity of leachate generated and the size of the aquifer. If the amount of leachate produced is large and the amount of ground water is small, for example, dilution may be minimal.

- Adsorption. As chemicals are transported through the soil column, they may concentrate at solid-liquid interfaces. Some chemicals, such as acetone, do not tend to partition to soil; for these chemicals, adsorption would not be expected to be a major mechanism for attenuation (Roy and Griffin 1985). Other chemicals, such as carbon tetrachloride, will adsorb more strongly to the soil; the movement of these chemicals toward the ground water would thus be retarded (Roy and Griffin 1987).

- Dispersion. Liquid molecules will tend to move through a porous medium at different velocities as a result of variations in pore size, density gradients, and other factors. This phenomenon is known as dispersion, and will tend to reduce the peak chemical concentrations in leachate.

- Biological and Chemical Degradation Chemicals may degrade as the result of biological and/or chemical processes as they are transported through the soil column. Chemical degradation generally involves certain hydrolysis reactions between the contaminant and water. Biological degradation is caused by micro-organisms which break down organic substances. The rate of degradation is highly variable; some chemicals, such as chloromethane, degrade readily, whereas others such as PCBs, are highly persistent (see, for example, Mabey et al. 1982).

The effect of these attenuation mechanisms is currently under evaluation, both by OWMC and by the general scientific community. For example, studies conducted for OWMC indicate that dilution is expected to result in approximately a three-fold reduction in the concentrations of contaminants reaching the aquifer (Gartner Lee 1987). Furthermore, investigations regarding the attenuation of organics in clay have also been commissioned by OWMC. The state of knowledge with respect to attenuation processes, however, is such that in most cases it is not possible to quantify the long term reductions in contaminant concentrations that may occur.

As previously noted, a very long time will be required for chemicals to travel from the landfill through the 15-20 meter clay layer to the bedrock aquifer. During this extended period of time, it is expected that biological and chemical degradation processes will occur, thereby reducing the concentrations of many contaminants, particularly organics. The impacts of these degradation processes have not been rigorously evaluated by OWMC due to gaps in the available data. It has been possible, however, to examine the potential effect of these processes on eventual concentrations in ground water by assuming degradation half-lives[1] for organic chemicals present in the leachate.

In the analysis conducted, low molecular weight organics and high molecular weight organics were examined separately. This approach was taken since the low molecular weight compounds considered are generally less environmentally persistent and would also pass through the clay more quickly than those with high molecular weights. The length of time required to reach the aquifer was assumed to be 1 000 years for low molecular weight organics and 100 000 years for high molecular weight organics, values which are consistent with estimates developed by the hydrogeology consultants to OWMC (Gartner Lee 1987).

To assess the impact of variations in degradation rates, two half-lives were assumed for both the low molecular weight and high molecular weight organics considered:

Case 1: Low molecular weight organics were assumed to have a half-life of 50 years; high molecular weight organics, a half-life of 5 000 years.

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[1] The "half-life" is defined in this section as the time required for half of the amount of the chemical to degrade.

Case 2: Low molecular weight organics were assumed to have a half-life of 250 years; high molecular weight organics, a half-life of 25 000 years.

The following equation was used to calculate contaminant concentrations in the aquifer at the site boundary:

$$\frac{C_a}{C_l} = \frac{\exp[-kt]}{D}$$

Where:

C_a = contaminant concentration in the aquifer, mg/L

C_l = contaminant concentration in the leachate, mg/L

k = degradation rate constant, yr^{-1}

t = travel time to the aquifer, yr

D = dilution in the aquifer, 3 (Gartner Lee 1987)

To estimate degradation rate constants using the assumed half-lives, the above equation can be rearranged, and the ratio (C_a/C_l) set equal of 0.5:

$$k = \frac{-\ln(0.5)}{t_{1/2}} = \frac{0.693}{t_{1/2}}$$

Where:

k = degradation of half-life, yr^{-1}

$t_{1/2}$ = degradation of half-life, yr

Based on this equation, the following degradation rate constants (k) were calculated:

Case 1: Low molecular weight organics: 0.0139 yr^{-1}
High molecular weight organics: 0.000139 yr^{-1}

Case 2: Low molecular weight organics: 0.00277 yr^{-1}
High molecular weight organics: 0.0000277 yr^{-1}

The results of the study, as summarized in Table 6-7, demonstrate that even relatively long degradation half-lives result in significant reductions in chemical concentrations in the aquifer. Based on the assumptions of Case 2, chemical concentrations in ground water would be approximately 50 times lower than the concentrations in leachate; in Case 1, chemical concentrations are reduced by over 3 000 000 fold.

It should be recognized that there is uncertainty in predicting degradation half-lives for the chemicals listed in Table 6-7. Data with respect to the rate at which the various attenuation mechanisms are likely to occur are largely unavailable. Furthermore, it is possible that the degradation byproducts may also be relatively toxic. The evaluation does suggest, however, that even very slow degradation processes that occur in the 15-20 meter clay layer beneath the landfill would lead to much lower chemical concentrations in the ground water than are initially present in the leachate. Research to more accurately quantify the attenuation mechanisms likely to limit any contamination of the aquifer is currently being performed by OWMC.

TABLE 6-7

EFFECT OF CHEMICAL AND BIOLOGICAL DEGRADATION ON CHEMICAL CONCENTRATIONS IN THE AQUIFER[1]

	Leachate Concentration (mg/L)	Ground Water Concentration (mg/L)	
		Case 1[2]	Case 2[3]
Low Molecular Weight Organics			
Trichloroethylene	4.7 E-02	1.5 E-08	1.0 E-03
Tetrachloroethylene	9.7 E-02	3.1 E-08	2.0 E-03
Toluene	8.6 E+00	2.7 E-06	1.8 E-01
Xylene	1.4 E-02	4.5 E-09	2.9 E-04
Styrene	9.0 E-03	2.8 E-09	1.9 E-04
Cresol	4.4 E-03	1.4 E-09	9.2 E-05
Ethanol	2.2 E+00	7.0 E-07	4.6 E-02
Petroleum Distillates	1.2 E-01	3.8 E-08	2.5 E-03
Dichlorodifluoromethane	5.8 E+00	1.8 E-06	1.2 E-01
Trichlorotrifluoroethane	1.2 E+01	3.7 E-06	2.4 E-01
Trichloroethane, 1,1,1	2.0 E-01	6.4 E-08	4.2 E-03
Benzene	1.7 E-02	5.4 E-09	3.6 E-04
Formaldehyde	6.9 E+01	2.2 E-05	1.4
Phenol	8.3 E-01	2.7 E-07	1.7 E-02
High Molecular Weight Organics			
Benzo(a)anthracene	1.6 E-04	5.1 E-11	3.3 E-06
Benzo(a)pyrene	7.0 E-05	2.2 E-11	1.5 E-06
PCDD/PCDF	7.7 E-09	2.4 E-15	1.6 E-10
PCBs	5.9 E-05	1.9 E-11	1.2 E-06
Naphthalene	3.2 E-03	1.0 E-09	6.7 E-05

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[1] All ground water concentrations assume a dilution of three-fold relative to leachate concentrations.

[2] Case 1: Low molecular weight organics: Travel time to the aquifer assumed to be 1 000 years. Degradation half-life assumed to to 50 years.
High molecular weight organics: Travel time to the aquifer assumed to be 100 000 years. Degradation half-life assumed to be 5 000 years.

[3] Case 2: Low molecular weight organics: Travel time to the aquifer assumed to be 1 000 years. Degradation half-life assumed to to 250 years.
High molecular weight organics: Travel time to the aquifer assumed to be 100 000 years. Degradation half-life assumed to be 25 000 years.

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CHAPTER 7

RELEASES ASSOCIATED WITH ON-SITE UPSETS AND ACCIDENTS

7.1 INTRODUCTION

The potential for on-site accidents or upsets exists at any industrial facility. Such events can be caused by equipment failure or operator error, and many result in the short-term release of potentially hazardous chemicals to the environment. A number of potential accident and upset scenarios that could occur at the proposed OWMC Facility have been identified by the engineering consultants to OWMC (see, for example, Monenco 1986). The following scenarios were quantitatively evaluated in the Phase 4A generic risk assessment (ENVIRON 1985):

- A tank farm fire;
- Mixing of incompatible wastes;
- Incinerator scrubber failure;
- pH upset during treatment of cyanide-containing wastes;
- pH upset during sulfide precipitation of heavy metals.

These events have been reexamined in the Phase 4B risk assessment. In addition, a detailed hazard analysis for the proposed facility is currently being conducted by consultants to OWMC. Those events identified as posing a potentially significant risk to the public health will be examined in detail during that study.

The first section of this chapter presents a general description of the releases associated with the upsets and accidents examined in the risk assessment. In the case of the tank farm fire, the methodology used to estimate the frequency of such an event is also discussed. Such estimates have not been developed for other on-site accident and upset scenarios, since the results of the Phase 4A generic risk assessment indicated that emissions associated with these events would not pose a significant risk to public health (ENVIRON 1985). The methodologies used to calculate emissions resulting from the on-site upset and accident scenarios and the frequency of a tank farm fire at the OWMC Facility are presented in detail in Appendix C.

The second section of this chapter describes the assessment of human exposure resulting from on-site upsets and accidents. This assessment consists of the following steps:

- Identification of exposure pathways for analysis in the risk assessment;
- Evaluation of the environmental transport and fate of the contaminants released; and
- Estimation of human exposure.

Details on the procedures used to estimate the magnitude of potential human exposures are provided in Appendices D and I.

7.2 RELEASE SCENARIOS

7.2.1 TANK FARM FIRE

Waste storage tanks will be provided at the OWMC Facility for the storage, blending, and pretreatment of pumpable wastes received from waste generators and from on-site waste sources. A review of the operating history of existing hazardous waste management facilities shows that the possibility exists for a fire to occur in the tank farm area (see, for example, Chemcontrol 1983). The potential public health risks associated with such an event were therefore evaluated in the risk assessment for the proposed OWMC Facility.

The analysis of a tank farm fire was based on a scenario involving the release of methylene chloride from a halogenated organic waste storage tank at the OWMC Facility. Methylene chloride was identified in the Phase 4A risk assessment as the most potentially hazardous chemical that may be released to the atmosphere in the event of a tank farm fire. Details on the selection of methylene chloride as the "surrogate" chemical for this analysis are presented in the Phase 4A generic risk assessment report (ENVIRON 1985).

Two scenarios involving a tank farm fire resulting in vaporization and subsequent release of methylene chloride to the atmosphere have been examined. General descriptions of the two scenarios are presented below.

7.2.1.1 Fire in the Halogenated Organic Waste Storage Tank Area

At the OWMC Facility, three halogenated organic waste storage tanks, each of 8 m^3 capacity will be located within a diked area (Monenco 1986). The tanks will be separated from each other by internal dikes. In the first release scenario, it was envisioned that a leak develops in a tank filled with a halogenated solvent waste (Battelle 1987). The spilled waste collects in the diked area immediately surrounding the tank, is ignited, and burns. The heat of the fire is sufficient to boil off the remaining contents of the tank, which are vented above the level of flame effect. For the purposes of the risk assessment, it was assumed that the atmospheric release consists entirely of methylene chloride, the surrogate chemical.

To provide a conservative estimate of the human health risks associated with a fire surrounding one of the halogenated organic waste tanks, it was assumed that the insulation for the storage tanks could be destroyed, and that the leak is sufficiently large to sustain a fire capable of vaporizing the remaining tank contents. It should be noted that the flammability of the selected surrogate chemical, methylene chloride, is relatively low. Therefore, the likelihood of a leak of methylene chloride igniting or resulting in a sustained fire is also low, although other chemicals which could also be present in the storage tanks may be more flammable. The scenario considered conservatively assumes that the liquid leak which causes the fire is composed of a relatively flammable chemical, whereas the vapor released consists solely of methylene chloride. The duration of the release has been estimated as approximately 2.6 hours, the period required to boil off an entire tank of methylene chloride (ENVIRON 1985). This estimate is conservative in that at least part of the contents of the tank would have to be spilled in order for the potential for a fire to exist.

7.2.1.2 Fire in the Organic Sludge Waste Area

In the second scenario examined, a leak or spill of organic sludge waste in the diked area adjacent to the halogenated organic waste area was considered (Battelle 1987). Sludge wastes will be contained in six tanks of 120 m^3 (31 700 gallon) capacity. Individual tanks will not be separated by dikes, although there will be a dike separating the organic sludge waste from the halogenated organic waste tanks.

In this scenario, it was envisioned that the entire 120 m^3 contents of one organic sludge tank is released in the diked area surrounding the tanks. The released material is ignited and burns. As shown in in Appendix C, the radiant heat flux to the halogenated organic waste tanks from the burning organic sludge would be sufficient to heat the contents of a tank to boiling, and thus result in a release to the atmosphere.

In estimating the risks associated with releases in this second scenario, it was assumed that the boiling tank would be full of a halogenated solvent waste. As in the first scenario, this waste was represented by the surrogate chemical, methylene chloride. It was again assumed that the insulation for the halogenated organic storage tanks would be destroyed, and that the leak from the sludge tank would be large enough to sustain a fire capable of vaporizing the contents of the halogenated organic tank. The estimated duration of release for this scenario is 31 to 37 minutes (Battelle 1987).

7.2.1.3 Frequency of a Tank Farm Fire

In the Phase 4A generic risk assessment (ENVIRON 1985), it was found that the level of human exposure associated with a tank farm fire could be significant, although not likely to cause incapacitating illness or death. Given these results, it was recognized that an estimate of the frequency of tank farm fires would be necessary to develop a more complete understanding of the hazards posed by these events. Therefore, a fault tree analysis was performed in Phase 4B to determine how often a tank farm fire might be expected to occur at the proposed OWMC Facility (Battelle 1987).

A tank farm fire causing (at least partial) boil-off of the contents of one of the halogenated organic waste tanks was represented as a sequence of two events:

- A leak or spill from a halogenated organic waste tank or an adjacent organic sludge waste tank; and
- Ignition of the spilled material.

Battelle used an event tree/fault tree analysis, combined with empirical spill ignition data, to estimate the frequency with which this sequence could occur. Assuming that all six organic sludge waste tanks and all three halogenated organic waste tanks are full, and that the tank groups are not segregated, the overall chance of a fire of sufficient intensity to volatilize the halogenated organic waste was estimated by Battelle (1987) to occur with a frequency not exceeding about one in 100 years. This value represents the combined frequency for the two scenarios examined.

It should be noted that the event tree/fault tree analysis did not take into account the mitigative measures which could reduce the hazard of ignition after the spill occurs. Such measures include:

- Detecting the spill before ignition and preventing immediate or delayed ignition;
- Fire suppression before vapor boil-off becomes significant.
- Using fireproof tank insulation; and
- Segregating groups of halogenated and sludge tanks.

Such mitigative measures will be an integral part of the facility's design and operation. Accordingly, Battelle has indicated that, in practice, a tank farm fire resulting in vapor boil-off would be expected to occur less frequently than once in 100 years. For example, if the individual halogenated waste tanks were to be segregated, the estimated frequency would drop to one in every 700 to 1 300 years.

7.2.2 MIXING OF INCOMPATIBLE WASTES

The accidental mixing of incompatible wastes at a hazardous waste facility can cause chemical reactions which may result in generation of toxic gases. The scenario considered in the risk assessment involved the mixing of strong acids with sulfide-containing wastes resulting in the generation of hydrogen sulfide gas. This scenario was developed in earlier work for OWMC on the selection of a technical buffer zone (M.M. Dillon 1984).

In estimating the magnitude of hydrogen sulfide emissions, it was assumed that operational and engineering controls would prevent mixing of acids and sulfide wastes in bulk quantities. Rather, as a worst-case, it was envisioned that a single 200-liter drum of 4 percent sulfuric acid was mixed with sulfide contaminated material in the physical/chemical treatment building. The gas produced by this reaction, hydrogen sulfide, is emitted first into the building and then exhausted through the ventilation system to the atmosphere. Release rates for this situation were estimated from laboratory simulations on a reduced scale (see M.M. Dillon 1984). Based on these release rates, the emission duration was estimated to be 42 minutes (ENVIRON 1985).

7.2.3 INCINERATOR SCRUBBER FAILURE

The proposed OWMC incinerator includes a three-stage scrubbing system to capture acid gases such as hydrogen chloride, hydrogen fluoride, sulfur dioxide, and nitrogen oxides generated during the combustion process. Effective control of acid gases released from the incinerator stack depends on the proper functioning of the scrubber system. In the event of an incinerator flue gas scrubber failure, elevated emissions of acid gases from the stack could occur.

To limit the duration of such elevated emissions, the design and operation of the proposed facility incorporates the following features in the event of a scrubber failure (Monenco 1987):

- Waste feed to the incinerator would be shut down immediately;
- Flue gas flow would continue, but would decrease to 10 percent of normal within four minutes;
- The emergency power system would resume normal water supply and scrubber liquid to the scrubbing system within thirty seconds following failure.

Thus, following the initial thirty seconds after scrubber failure, it is expected that contaminant concentrations in the emitted gases would return to those during normal scrubber operation.

Emission rates for the acid gases during the thirty seconds of scrubber failure were estimated from predicted flue gas contaminant concentrations supplied by Monenco (1987). To calculate the mean flow for the thirty second failure period, the decrease in volumetric gas flow to 10 percent of normal was assumed to be linear over the four minute period.

7.2.4 CYANIDE TREATMENT - UNCONTROLLED pH

Cyanide bearing wastes received at the OWMC Facility will be destroyed by chemical oxidation in an enclosed batch reactor in the physical/chemical treatment plant. Under normal operating conditions, air will infiltrate into the reactor due to a vacuum created in the vent system. It has been conservatively assumed that this air has a hydrogen cyanide concentration consistent with an equilibrium being established with the reaction solution. Cyanide vapor-laden air will be vented through a scrubber with an efficiency of 90 percent.

Under upset conditions, it was assumed that the pH control system fails, allowing the pH of the reaction mixture to fall below the normal operating range. At a lower pH, the initial concentration of hydrogen cyanide vapor in equilibrium with the solution would be higher than under normal conditions. The increased hydrogen cyanide releases corresponding to the drop in pH were examined in the risk assessment for the OWMC Facility. It was assumed that the absorber efficiency remains 90 percent during the upset scenario.

7.2.5 SULFIDE TREATMENT - UNCONTROLLED pH

The removal of heavy metals from waste solutions at the OWMC facility may involve a two-step precipitation process which includes the addition of a sodium sulfide solution to the waste under alkaline conditions. This results in the precipitation of heavy metals as sulfides. Under normal operating conditions, emission of hydrogen sulfide is negligible.

If, however, the pH is allowed to fall too low during abnormal operating conditions, the concentration of gaseous hydrogen sulfide in equilibrium with the waste solution would increase. Under these conditions, the release of hydrogen sulfide gas to the atmosphere would be expected to increase. In the analysis of this event, it was assumed that air saturated with hydrogen sulfide is vented through a water scrubber with a removal efficiency of 90 percent.

7.3 ESTIMATION OF POTENTIAL HUMAN EXPOSURES

The potential human health risks associated with short-term emissions resulting from on-site upsets and accidents can be classified as follows:

- Acute health risks resulting from short-term exposures.
- Chronic health risks resulting from short-term exposures. For example, an exposure of limited duration to a carcinogen could conceivably contribute to the lifetime cancer risk.
- Chronic health risks resulting from long-term exposures that are associated with a single release. For example, the release of a persistent chemical, such as PCBs, could lead to long-term soil contamination and the potential for chronic exposures.

Since OWMC is committed to taking those remedial measures required to prevent unacceptable long-term exposures resulting from upsets and accidents, only short-term exposures have been considered in the risk assessment. These acute exposures are all based on direct inhalation of contaminants since each of the on-site accident and upset release scenarios examined results in a short-term emission to the atmosphere. Furthermore, the assessment was limited to an evaluation of acute health risks, since the extrapolation of chronic toxicity data, such as those for cancer, to exposure periods of only a few hours or less was judged not to be appropriate for the chemicals examined.

Ambient air concentrations associated with short-term emissions were estimated in the risk assessment based on dispersion modeling performed by the Ontario Research Foundation (ORF 1985). In the model, fumigation conditions were assumed for releases from the incinerator stack; a ground level inversion was assumed for the other short-term emissions.[1] These conditions represent the "worst case" in estimating off-site chemical concentrations in air.

In applying the dispersion models, different time-averaging periods were assumed in analyzing the short-term emission scenarios:

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[1] During a ground level inversion, a layer of relatively stable air is formed at or close to ground level; emissions into that layer will be poorly dispersed due to a lack of turbulence. Fumigation conditions are those that exist when emissions are initially released into a inversion layer aloft. Emissions can later be dispersed to ground level in relatively high concentrations as an unstable layer, growing upwards from the ground, intersects with the plume aloft.

- For emissions resulting from a tank farm fire, mixing of incompatible wastes, the treatment of cyanide wastes, and the treatment of heavy metal wastes, maximum one hour concentrations were used to represent acute chemical exposures.
- For emissions resulting from failure of the incinerator scrubber, a maximum two-minute average concentration was used to characterize exposure.

The off-site chemical concentrations in air calculated using these modeling assumptions are presented in Appendix D.

To assess the magnitude of exposures to the chemicals in air, certain assumptions were made with respect to the location of the human population and the period of exposure. For example, all exposures were based on an individual located at the point of maximum off-site ground-level contaminant concentrations. This location varies according to the height of the release above ground level. In addition, exposures were assumed to occur for the entire duration of the release. These assumptions are likely to overestimate the level of human exposure, and thus form a conservative basis for risk assessment.

For releases associated with a tank farm fire, human health risks were estimated by calculating the systemic dose received over the maximum period of exposure (2.6 hours). This procedure was used as a result of the systemic toxicity associated with methylene chloride. For the other upset and accident events considered, predicted concentrations in air were simply compared to the maximum concentrations not believed to have the potential to produce an observable effect in an exposed individual. This technique for evaluating potential risks was deemed appropriate since the chemicals released in these scenarios are expected to cause localized irritation rather than systemic toxicity.

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CHAPTER 8

RELEASES ASSOCIATED WITH OFF-SITE TRANSPORTATION ACCIDENTS

8.1 INTRODUCTION

Wastes, additives, and reagents will be transported to the proposed OWMC Facility from the Queen Elizabeth Way (QEW) along the preferred access route (Regional Road 24, King's Highway 20, and Shram Road South). Accidents and malfunctions involving vehicles carrying these materials may result in spills along the access route.

In the Phase 4A generic risk assessment (ENVIRON 1985), it was concluded that accidents along the local access roads would be of greatest concern due to the proximity of residences and because the relative concentration of OWMC-related traffic on these roads will be higher than along the Provincial highway system, i.e., the 400 Series routes and the QEW. Furthermore, it was determined in Phase 4A that the risks associated with spills of hazardous wastes would be significantly greater than those posed by spills of additives and reagents. For these reasons, the Phase 4B risk assessment has been restricted to an analysis of spills of hazardous waste along the local access route.

Contaminant releases associated with two transportation-related spill scenarios have been examined in the risk assessment. These two scenarios were:

- A spill onto a roadway, with subsequent release of chemicals to the atmosphere. This scenario was previously evaluated in the Phase 4A risk assessment (ENVIRON 1985).
- A spill into a body of surface water adjacent to the local access route. This scenario was not evaluated in Phase 4A.

This chapter presents a general description of the approaches used to (1) characterize the nature, magnitude, and frequency of the releases associated with these two spill scenarios, and (2) assess the potential magnitude of resulting human exposure. Details of these methodologies are provided in Appendices C, D, E, and I.

8.2 SPILL ONTO A ROADWAY

8.2.1 RELEASE SCENARIO

The first transportation accident scenario considered assumes that a 18 tonne tank truck carrying a load of liquid waste is involved in a collision on the local access road, resulting in a tank rupture and a spill onto the roadway. The released liquid is assumed to spread across the road surface to form a pool from which volatile constituents are released to the atmosphere. The pool is assumed to be circular with the center of the circle coincident with the centerline of the road, and with the diameter constrained by the width of the paved surface.

For the purposes of the risk assessment, the spilled liquid was assumed to consist entirely of methylene chloride, which was identified in the Phase 4A analysis as the most potentially hazardous chemical that could be released in this scenario (ENVIRON 1985). The selection of methylene chloride as an appropriate surrogate was based on a ranking scheme using the following factors: (1) the acute toxicity of the chemical; (2) the potential for the chemical to be released to the atmosphere; and (3) the quantity of chemical available for release. The surrogate selection methodology used in the analysis is presented in Appendix B.

In the volatilization model used to estimate the rate of release of methylene chloride to the atmosphere, the most critical chemical-specific parameter is vapor pressure. The estimated emission rate from the pool of liquid is sensitive also to certain environmental parameters, including wind speed and ambient temperature. For the purposes of the risk assessment, it was assumed that the ambient temperature is 25°C, which is typical of summertime temperatures in Southern Ontario and would overestimate the rate of chemical vaporization that would occur during the winter months. For example, the emission rate at 0°C would be approximately three times lower than at 30°C. Details on the volatilization model used in this calculation are given in Appendix C.

8.2.2 THE FREQUENCY OF A SPILL ONTO THE LOCAL ACCESS ROAD

The frequency of a truck accident-related spill of special wastes along the local access route was provided by the transportation consultants to OWMC (M.M. Dillon 1986). These frequency estimates were based on the general vehicle accident data compiled for all classes of vehicles along the local and regional access roads (M.M. Dillon 1986). To estimate spill frequencies for the risk assessment, it was necessary to use additional data concerning the ratio of the accident frequency for trucks to that for all vehicles, and the frequency with which truck accidents result in spills. These data were derived from studies conducted by Pilorusso (1983) and M.M. Dillon (1986).

Based on information corresponding to a facility capacity of 150 000 t/a, a spill associated with a truck accident was estimated to occur along the local access route about once in

10.5 years. This estimate applies to a spill involving any special waste being transported to the OWMC Facility, rather than just those wastes with the potential to produce adverse health effects in the exposed population. It should be recognized that a spill of pumpable waste is predicted to occur approximately once every 14 years. Furthermore, it was estimated in the Phase 4A Generic Risk Assessment that only 8 percent of the special waste transported to the facility would have the potential to cause adverse effects in the exposed off-site population in the event of a spill onto the roadway (ENVIRON 1985). For these spills, the estimated frequency would be reduced to once in 132 years. Furthermore, it has been estimated that only 0.8 percent of the special waste to be received by the OWMC Facility would consist of methylene chloride, the most hazardous special waste (ENVIRON 1985). The spill frequency for the most hazardous waste received at the OWMC Facility would therefore be reduced to approximately once in 1 320 years.

Each of these predicted spill frequencies correspond to a facility capacity of 150 000 t/a. At the ultimate facility capacity of 300 000 t/a, the number of truck shipments of special wastes would double, so that estimated spill frequencies would also be expected to double. Details of the methods used to estimate the frequency of a spill onto the roadway are provided in Appendix C.

8.2.3 EVALUATION OF POTENTIAL HUMAN EXPOSURE

In evaluating potential public health risks resulting from a spill onto a roadway, the most significant exposure pathway was considered to be direct inhalation of chemicals volatilizing from the road surface.

In this spill scenario, the concentration of chemicals in air was calculated using a model that predicts dispersion in the atmosphere. In applying the model, it was assumed that the area in the vicinity of the spill location is essentially flat, and that no major perturbations are caused by the proximity of tall buildings or other topographic features. "Worst case" atmospheric conditions (Stability Class F and a wind speed of 1 meter per second) were assumed in the calculations. These atmospheric stability conditions are conservative in that they maximize predicted chemical concentrations in air.

The dose received by an exposed individual will be directly proportional to chemical concentration in air. In general, the chemical concentration in air will decrease as the distance from the spill increases. In the risk assessment, exposures were conservatively evaluated at 20 meters, the shortest estimated distance between the centerline of the road and a potential receptor (M.M. Dillon 1986). The concentration of methylene chloride in air at this point was estimated using the approach shown in Appendix D.

The dose received will also be directly proportional to the duration of exposure, which will be determined by one of three factors: (1) the time needed for evacuation of potentially exposed individuals from the vicinity of the spill, (2) the time required for cleanup of the spill by emergency response personnel, or (3) the time required for the entire spill to volatilize. Since the amount of time necessary for the entire spill to volatilize is likely to be relatively long, it was assumed that the period of exposure would be governed by the time required for either evacuation or cleanup. These responses will be influenced by certain variables, such as the time and location of the spill, the rapidity of spill reporting, and the placement of emergency services.

Due to the uncertainties in predicting emergency response time, estimates of the duration of exposure range from several minutes to several hours. For the purposes of the risk assessment, the period of exposure was assumed to be one hour. This was considered a conservative assumption due to the close proximity of potential spill sites to both the proposed OWMC Facility, where response personnel and equipment will be located, and to regional and municipal emergency facilities.

8.3 SPILL INTO SURFACE WATER

8.3.1 RELEASE SCENARIO

The local access route to the proposed OWMC Facility crosses several streams, including Twenty Mile Creek, Sixteen Mile Creek, and Fifteen Mile Creek. A transportation accident which occurs in proximity to any of these crossings could result in a spill of potentially hazardous waste into the stream. In the risk assessment, Twenty Mile Creek was selected for evaluation since has a number of sensitive downstream uses and is the largest stream along the preferred access route.

Bulk liquids, solids and sludges, and containerized waste will be transported to the proposed facility along the local and regional roads in trucks of varying types and sizes. In the risk assessment, a tank truck carrying a load of bulk liquid waste was envisioned to be involved in a spill incident at the intersection of Regional Road 24 and Twenty Mile Creek, resulting in a partial release of the tank truck contents directly into the creek. This spill scenario is likely to provide a conservative estimate of contaminant concentrations in surface water since: (1) a spill directly into the stream maximizes the quantity of liquid entering the water; and (2) a spill of bulk liquids provides less opportunity for spill containment and control relative to bulk sludges and solids, or to containerized waste.

In the examination of the spill onto the roadway, only those releases resulting from collisions were considered. In evaluating a spill into Twenty Mile Creek, however, releases associated with both collisions and equipment malfunctions were considered. Spills caused by equipment malfunctions may be expected to involve smaller quantities of waste, but may occur more frequently than those caused by transportation accidents.

Estimates of the potential magnitude of the spill were based on data taken from computer records maintained by the U.S. Department of Transportation (USDOT) Office of Hazardous Materials Transportation. Three spill sizes were chosen from the USDOT data base for the current analysis: 230 liters (approximately 50% of the reported spills were greater than or equal to 230 liters), 1 500 liters, (25% of the reported spills were greater than or equal to 1 500 liters), and 19 000 liters, (5% of the reported spills were greater than or equal to 19 000 liters).

Consideration was also given to the chemical and physical properties of the possible constituents of the spilled waste, particularly those properties which influence chemical concentrations in the stream. Three types of chemicals were identified as having significantly different transport properties if spilled into surface water: "mixers", chemicals which would be expected to mix readily with the water;

"sinkers", which would sink to the bottom of the stream; and "floaters", which would float along the water surface. In the risk assessment, three separate analyses were conducted to examine the impacts of spills consisting entirely of each of the three chemical types.

8.3.2 THE FREQUENCY OF A SPILL INTO TWENTY MILE CREEK

The frequency with which a transportation spill into Twenty Mile Creek would be expected to occur was evaluated using data provided by M.M. Dillon (1986) and an approach similar to that described in Section 8.2.2 for spills onto a roadway. The frequency of a truck spill (of any size) occurring along the section of the local access route intersecting Twenty Mile Creek was estimated to be 0.04 spills per year, or one spill in every 25 years. The frequency with which these spills could enter Twenty Mile Creek was estimated from the ratio of the length of road near the stream (i.e., the length of the bridge and the immediately adjacent sections of the roadway) to the length of the section of the access route containing the bridge. On this basis, a spill near the bridge over Twenty Mile Creek with the potential to enter the stream directly was estimated to occur approximately once in 435 years. It should be noted that the frequency of a spill of pumpable waste into Twenty Mile Creek would be expected to be even lower.

As indicated in Section 8.3.1, three different spill sizes were chosen for analysis in the risk assessment. By combining the frequency of a spill occurring near the bridge crossing and the frequency of a release of a certain size, the frequency of a release of certain size spill near the bridge can be estimated. These frequencies are summarized in Table 8-1. As shown, spills of 230 liters (or less), 1 500 liters (or less), and 19 000 liters (or less) were estimated to occur (on average) about once in 875 years, 1 750 years, and 8 750 years, respectively.

At the ultimate facility capacity of 300 000 t/a, the estimated frequency of spills into Twenty Mile Creek would double. Thus spills of 230 liters (or less), 1 500 liters (or less), and 19 000 liters (or less) are estimated to occur once in 440 years, 875 years, and 4 400 years, respectively. Details of the calculations of the frequency of a spill into Twenty Mile Creek are provided in Appendix C.

8.3.3 EVALUATION OF POTENTIAL HUMAN EXPOSURE

The analysis of of potential human exposure to chemicals released into Twenty Mile Creek involved the following steps:

- Characterization of the surface water at the time the spill occurs,
- Prediction of contaminant concentrations in the surface water, and
- Estimation of human dose.

TABLE 8-1

FREQUENCY OF A SPILL OCCURRING NEAR TWENTY MILE CREEK

Size of Spill (liters)	Frequency of a Spill Occurring Near Twenty Mile Creek (a^{-1})	Frequency of Specific Size Spill Occurring	Frequency of Specific Size Spill Occurring Near Twenty Mile Creek (a^{-1})	Recurrence Interval of a Specific Size Spill Occurring Near Twenty Mile Creek (a)
230	0.0023	0.50	0.0011	875
1 500	0.0023	0.25	0.00057	1 750
19 000	0.0023	0.05	0.00011	8 750

The following sections of this chapter outline the methodology used in this analysis. Details of the analysis are presented in Appendix E.

8.3.3.1 Characterization of Twenty Mile Creek

From its source located near the city of Hamilton, Twenty Mile Creek meanders eastward in a shallow valley. The creek crosses beneath Regional Road 24, which is part of the preferred local access route, before reaching the escarpment at Ball's Falls, where it drops approximately 26 meters into a narrow gorge. At this point, the creek turns northwards and flows across a plain to a large bay on Lake Ontario at Jordan Harbor. The distance from Regional Road 24, the point considered in the spill scenario, to this bay is approximately 6.4 kilometers. This section of the creek has been modeled in the risk assessment.

In the risk assessment, the median flow of Twenty Mile Creek was chosen to represent the flow condition at the time of the spill. In order to estimate this flow, monthly and daily discharge records from the gaging station located at Ball's Falls were analyzed (Water Survey of Canada 1985). By comparing the monthly average flows of the years 1976 to 1981 to the monthly average flows of the years 1957 to 1984, it was determined that 1978 represented the year when the flow was nearest to an average flow or typical flow year (see

Appendix E). On this basis, the daily discharge records for the year 1978 were analyzed to determine the median flow for the year.

8.3.3.2 Prediction of Contaminant Concentrations

As outlined in Section 8.3.1, chemicals which spill into water can be classified into three general categories: 1) "mixers", soluble compounds that mix readily with water; 2) "floaters", immiscible (slightly soluble or insoluble) compounds that float on water; and 3) "sinkers", immiscible compounds that sink to the bottom of a watercourse (Thibodeaux 1980). To account for the variability of the composition of the wastes to be transported to the proposed OWMC, one chemical from each of the above categories was selected for analysis. The parameters used in selecting surrogates to represent each chemical category were: (1) acute toxicity, and (2) quantity of chemical available for release. Based on these two criteria, pyridine was chosen as the "mixer"; petroleum distillates was chosen as the "floater"; and methylene chloride was chosen as the "sinker." The surrogate selection procedure used in the analysis is described in detail in Appendix B of this report.

A series of environmental transport models were used to characterize the behavior of the three types of chemicals in water. These models incorporate the following assumptions:

- The "mixer", pyridine, was assumed to mix rapidly with the stream water. Dilution and dispersion of pyridine in the water were estimated to predict chemical concentrations at various locations downstream from the access road crossing.
- The "sinker", methylene chloride, was assumed to sink and form a separate liquid phase at the bottom of the stream. The rate of dissolution of methylene chloride from this organic phase was estimated to predict the resulting concentrations in water.
- The "floater", petroleum distillates, was assumed to form a separate phase on the water surface. The rate of volatilization of petroleum distillates from Twenty Mile Creek to the atmosphere was used to predict the removal from the stream as the chemical is transported downstream. No mixing into the water was assumed.

Although these models do not consider all the processes that govern the fate and transport of pollutants in the environment, the assumptions made in applying the models are conservative in that they will tend to overstate the extent of contamination under foreseeable circumstances. Based on these assumptions, the pyridine and methylene chloride concentrations in Twenty Mile Creek were calculated and are shown in Table 8-2. The distances required for complete volatilization of the petroleum distillates from the creek are presented in Table 8-3. The models and assumptions used in the analyses are described in Appendix E.

8.3.3.3 Estimation of Human Dose

In the evaluation of a spill of special waste into Twenty Mile Creek, ingestion was identified as the most significant exposure pathway. It should be noted, however, that no

TABLE 8-2

CONCENTRATIONS OF PYRIDINE AND METHYLENE CHLORIDE IN TWENTY MILE CREEK

	Concentration (mg/l)
Methylene Chloride	9.6
Pyridine	
• 230 l spill	
– 100 m downstream	541
– 3 000 m downstream	99
– 6 400 m downstream	68
• 1 500 l spill	
– 100 m downstream	3 609
– 3 000 m downstream	659
– 6 400 m downstream	451
• 19 000 l spill	
– 100 m downstream	45 112
– 3 000 m downstream	8 236
– 6 400 m downstream	5 639

TABLE 8-3

DISTANCE REQUIRED FOR VOLATILIZATION OF PETROLEUM DISTILLATES FROM TWENTY MILE CREEK*

Spill Size (liters)	Distance (meters)
230	951
1 500	1 737
19 000	4 038

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* The distances calculated do not take into consideration the increased volatilization losses which could occur as the stream passes over Balls Falls.

drinking water intakes have been reported in Twenty Mile Creek downstream from the access road bridge. For this reason, it was assumed in the risk assessment that an individual located at the edge of Twenty Mile Creek takes water directly from the stream. The amount of water ingested by this individual has been assumed to be one-fourth the average daily consumption of water; 0.5 l for an adult (USEPA 1980) or 0.25 l for a child (USEPA 1980).

Due to the effects of dilution and dispersion, concentrations of the mixer, pyridine, in Twenty Mile Creek were predicted to decrease as contamination moves downstream toward Jordan Harbor. Therefore, the levels of exposure associated with ingestion of pyridine-contaminated water taken at various distances from the access road crossing were evaluated in the risk assessment. For the sinker, methylene chloride, only concentrations near the site of the spill were evaluated since it was predicted that the effects of dilution and dispersion would not have a significant impact on the downstream chemical concentrations. The predicted doses associated with ingestion of stream water contaminated with pyridine and methylene chloride are summarized in Table 8-4. Ingestion of the floater, petroleum distillates, was not evaluated since it was assumed that an individual would not drink water from a visibly contaminated stream.

TABLE 8-4

PREDICTED HUMAN DOSES FOR PYRIDINE AND METHYLENE CHLORIDE

	Dose (mg/kg)* Adult	Dose (mg/kg)* Child
Methylene Chloride	0.069	0.077
Pyridine		
• 230 liter spill		
– 100 m downstream	3.9	4.4
– 3 000 m downstream	0.71	0.80
– 6 400 m downstream	0.49	0.55
• 1 500 liter spill		
– 100 m downstream	25.8	29.1
– 3 000 m downstream	4.7	5.3
– 6 400 m downstream	3.2	3.6
• 19 000 liter spill		
– 100 m downstream	322	363.8
– 3 000 m downstream	58.8	66.5
– 5 400 m downstream	40.3	45.5

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* Based on ingestion of 0.5 liter of water by a 70 kg adult, or 0.25 liter of water by a 31 kg child.

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CHAPTER 9

ASSESSMENT OF THE TOXICITY OF THE CONTAMINANTS OF CONCERN

9.1 INTRODUCTION

The study of toxicology deals with the relationships between exposure to chemical substances and the potential for adverse human health effects. While chemical substances are often regarded as either "safe" (non-toxic) or "unsafe" (toxic), this general classification is misleading since all chemicals can cause biological injury, disease, or death under certain conditions of exposure.

Chemical exposure can be classified as either chronic (long-term) or acute (short-term). Chronic exposures usually occur to small doses of a chemical and do not produce immediately observable effects. However, acute exposures are usually associated with high doses of a chemical, and may produce immediately observable effects. These effects may cease when the exposure ceases (i.e., acute affects) or they may persist beyond the period of exposure (i.e., chronic effects associated with acute exposure). The assessment of the chronic and acute toxicity of the chemicals examined in the risk assessment is described in this chapter.

The Phase 4A Generic Risk Assessment report (ENVIRON 1985a) included detailed toxicity profiles for the chemicals of concern. While revised profiles have not been prepared in Phase 4B, the toxicity data used in Phase 4A has been updated to include a consideration of all currently available information.

9.2 CHRONIC TOXICITY

9.2.1 INTRODUCTION

The toxic characteristics of a chemical may be categorized according to the systems they affect (e.g. liver, kidney, central nervous system) or the types of toxicity they cause (e.g. cancer, birth defects). In assessing chronic exposures, chemicals are divided into two categories: carcinogens (chemicals which may cause cancer) and non-carcinogens. This distinction is made because of the difference in methods typically used to evaluate the risks associated with chronic exposure to carcinogens and non-carcinogens. For non-carcinogens, it is usually assumed that a minimum (threshold) dose exists which must be exceeded before a toxic effect is observed. For carcinogens, however, it is generally assumed that no such threshold exists, i.e. some risk is associated with any level of exposure. It should be noted that the "no-threshold" assumption may not be appropriate for certain chemical carcinogens.

9.2.2 CHRONIC EXPOSURE TO NON-CARCINOGENS

For non-carcinogenic chemicals, it is generally accepted that toxic effects will not be induced by sufficiently low doses. This assumption is supported by experiments that indicate a certain minimum number of molecules of a non-carcinogenic substance must reach and interact with some component of a cell before an adverse effect is produced. The minimally effective dose of any non-carcinogen that evokes a toxic response is called the threshold dose.

Safe exposure levels for non-carcinogens are typically determined by first identifying the dose at which no adverse effect is observed in an exposed population. This dose, known as the "no-observed-adverse-effect-level" (NOAEL), approximates a threshold dose for the population under study. Safe chronic exposure levels for non-carcinogens can be derived from human or laboratory animal NOAELs. However, NOAELs for chronic exposure are most commonly derived from animal studies due to limitations in available human exposure data.

It should be noted that not all effects of exposure to a chemical are adverse to health. Thus, a distinction is often made between a no-observed-adverse-effect-level (NOAEL) and a no-observed-effect-level (NOEL). For chronic exposures, a NOAEL and a NOEL are usually considered equivalent.

Although non-carcinogens are believed to operate by a threshold mechanism, it is not always possible to identify a NOAEL or NOEL from available studies. In these cases, all examined exposures produce an effect, so that only a lowest-observed-effect-level (LOEL) or a lowest-observed-adverse-effect-level (LOAEL) can be determined. When data corresponding to a NOEL or NOAEL are not available, a LOEL or LOAEL can be used to derive a safe exposure level.

The NOAEL determined from animal studies or observations in limited human populations is usually not considered acceptable as a safe limit for the general population. This is due to possible differences in susceptibilities between the test population and the general human population. Instead, NOAELs are used to calculate an Acceptable Daily Intake (ADI) by application of a safety factor such that:

$$\text{ADI} = \frac{\text{NOAEL}}{\text{Safety Factor}}$$

Safety factors used in deriving ADI values have been developed by various regulatory agencies. For instance, in the United States, the National Research Council (1977), the USEPA (1980), and the California Department of Health Services (1985) have recommended safety factors of 10, 100, or 1 000, depending on the source of data for the NOAEL. The following guidelines have been established for selecting the appropriate factor:

- A safety factor of 10 is used when the NOAEL is based on chronic human data. This factor is meant to account for intraspecies variability.
- A safety factor of 100 is used when the NOAEL is based on chronic animal data. This factor is meant to account for both intra- and interspecies variability.
- A safety factor of 1 000 is used when the NOAEL is based on subchronic animal data. This factor incorporates uncertainty in extrapolating from subchronic to chronic exposures and includes the two previous uncertainty factors.

In cases where a NOAEL has not been identified, the lowest-observed-adverse-effect level (LOAEL) can be used to estimate the ADI. In such cases, an additional safety factor, generally a factor of 10, is included.

Whether based on a NOAEL or a LOAEL, the concept of applying a safety factor to an observed threshold is predicated on the idea that the ADI sets a "safe" level. ADIs for those chemicals believed to operate via a threshold mechanism are presented in Table 9-1.

TABLE 9-1

CHRONIC TOXICITY DATA USED IN THE PHASE 4B RISK ASSESSMENT
NON-CARCINOGENS

Chemical	ADI (mg/kg-d)	Basis	References
Organics			
Acetone	2.86	EPA ADI based on TLV associated with minor irritation. Target organ: respiratory tract.	USEPA 1984b
Bromodichloromethane	0.006	Target organ: all.	USEPA 1986a
Bromoform	0.1	ENVIRON calculated ADI based on TLV. Target organ: liver.	ACGIH 1986
Chlorobenzene	0.03	Unpublished by EPA due to "last minute information." Target organs: all.	USEPA 1986a
Chloroform**	0.01	EPA ADI based on dog chronic oral study. Target organ: liver	USEPA 1986a
Cresols	0.05	EPA based on TLV. Target organs: blood, lungs.	USEPA 1986a
Dichlorobenzene	0.09	EPA based on rat subchronic oral NOEL. Target organs: all.	USEPA 1986a
Dichlorodifluoromethane	0.2	EPA based on rat chronic oral study yielding reduced body weight. Target organs: all.	USEPA 1986a
Ethanol	0.0495	ENVIRON calculated based on animal chronic inhalation study. Target organ: liver.	ENVIRON 1985a
Ethylene glycol	1.0	Rat 2-year oral NOEL. Target organ: kidney	ENVIRON 1985a
Formaldehyde*	0.012	ENVIRON calculated ADI based on rat chronic inhalation study. Target organs: all.	ENVIRON 1985a
Glycerol	0.017	Threshold Limit Value (TLV)	ENVIRON 1985a
n-Hexane	5.21	ENVIRON calculated from mouse 1-year NOEL. Target organs: all.	ENVIRON 1985a
Isopropyl alcohol	0.064	Human 6-week oral NOEL. Target organs: all.	ENVIRON 1985a
Kerosene	0.024	NIOSH recommended standard. Target organs: all.	ENVIRON 1985a
Methylene chloride**	0.06	EPA ADI based on rat chronic oral study. Target organ: liver.	USEPA 1986a
Methyl ethyl ketone	0.05	EPA ADI based on rat subchronic inhalation NOEL. Target organ: reproduction.	USEPA 1986a
Napthalene	0.0053	ENVIRON calculated ADI based on mouse subchronic study. Target organ: blood.	ENVIRON 1985a
O-Nitrophenol	0.075	ENVIRON calculated ADI based on analogy from subchronic mouse study with p-nitrophenol. Target organ: all.	GSRI 1981

TABLE 9-1 (continued)

CHRONIC TOXICITY DATA USED IN THE PHASE 4B RISK ASSESSMENT
NON-CARCINOGENS

Chemical	ADI (mg/kg-d)	Basis	References
Organics (continued)			
Petroleum Distillates	0.1	ENVIRON calculated ADI based on monkey inhalation LOEL. Target organ: CNS	ENVIRON 1985a
Phenol	0.1	EPA ADI based on rat subchronic oral LOAEL. Target organs: liver and kidney.	USEPA 1986a
Pyridine	0.002	EPA ADI Based on rat subchronic to chronic inhalation LOEL. Target organ: liver.	USEPA 1986a
Styrene	0.2	EPA ADI based on dog subchronic oral study. Target organs: liver and blood.	USEPA 1986a
Tetrachloroethylene**	0.02	EPA ADI based on rat subchronic oral study. Target organs: liver and kidney.	USEPA 1986a
Tetraethyl lead	1.0×10^{-7}	EPA ADI based on rat subchronic oral study. Target organs: liver and CNS.	USEPA 1986a
2,3,7,8-TCDD**	1.0×10^{-8}	OMOE derived ADI. Target organ: reproduction.	OMOE 1985
Toluene	0.3	EPA based on rat chronic inhalation NOAEL. Target organ: liver.	USEPA 1986a
1,1,1 Trichloroethane	0.029	ENVIRON calculated ADI based on mouse subchronic use inhalation study. Target organ: liver.	McNutt 1975
Trichlorofluorocarbon	0.30	EPA ADI. Target organs: all.	USEPA 1986a
Trichlorotrifluoroethane	30	EPA ADI. Target organs: all.	USEPA 1986a
Xylene	1.03	ENVIRON calculated ADI based on mouse teratology study. Target organ: reproduction.	Marks *et al*., 1982; Mirkova *et al*., 1982
Inorganics			
Cyanide	0.02	EPA ADI based on rat chronic oral study associated with myelin degeneration and thyroid effects. Target organs: CNS, thyroid.	USEPA 1986a
Fluoride	0.06	EPA ADI based on human oral epidemiologic data associated with objectionable dental fluorosis. Target organ: teeth.	USEPA 1986a
Nitrate	1.0	EPA ADI based on human infant chronic oral data associated with methemoglobinemia. Target organ: blood.	USEPA 1986a

TABLE 9-1 (continued)

CHRONIC TOXICITY DATA USED IN THE PHASE 4B RISK ASSESSMENT NON-CARCINOGENS

Chemical	ADI (mg/kg-d)	Basis	References
Acid Gases			
Hydrogen cyanide	0.02	EPA ADI based on rat chronic oral NOAEL. data. Target organs: CNS, thyroid.	USEPA 1986a
Nitrogen oxides	1.0	EPA ADI based on human infant chronic oral data. Target organ: blood.	USEPA 1986a
Metals			
Antimony	0.0004	EPA ADI based on rat chronic oral study associated with changes in blood glucose and chloresterol levels. Target organ: blood	USEPA 1986a
Arsenic*	0.0004	EPA ADI based on rat chronic oral study. Target organ: blood.	USEPA 1986a
Barium	0.05	Target organ: blood	USEPA 1986a
Beryllium	0.0005	EPA ADI based on rat chronic oral study. Target organ: all.	USEPA 1986a
Cadmium*	0.00075	Target organ: blood.	ENVIRON 1985a
Chromium VI*	0.005	Target organs: liver and blood.	USEPA 1986a
Copper	0.0286	ENVIRON calculated ADI based on NAS lower "safe and adequate" limit for adults. Target organs: liver and blood.	ENVIRON 1985a
Lead	0.004	EPA ADI based on human estimated oral chronic NOAEL for sensitive individuals. Target organ: blood.	USEPA 1984b
Mercury	0.002	EPA ADI based on rat chronic oral LOEL associated renal damage. Target organ: kidney.	USEPA 1984b
Nickel*	0.015	Target organs: reproduction and blood.	ENVIRON 1985a
Zinc	0.2	EPA ADI based on human chronic oral NOAEL from treatments of zinc sulfate. Target organs: reproduction and blood.	USEPA 1984b

* Carcinogenic through inhalation

** Treated as a carcinogen in the risk assessment. ADI values used to examine potential non-carcinogenic effects.

9.2.3 CHRONIC EXPOSURE TO CARCINOGENS

Unlike non-carcinogens, it is usually assumed that there is some risk associated with any level of exposure to a carcinogen and that the risk increases with increasing exposures. This assumption is based on an understanding of the mechanism by which cancers are thought to be produced.

Cancer is a term used to characterize the abnormal, uncontrolled growth of cells in animals or in humans. Carcinogenesis--the process that produces a cancer--appears to be a multi-stage process requiring an initiating event (usually considered to be a genetic alteration, or a "mutation-like" event, in a cell) followed under some conditions by proliferation of the affected cells into a tumor. The first type of event is called "initiation". Subsequent events, which are necessary for a cancer to be produced, can be facilitated by agents known as "promoters", substances that can transform initiated cells into cancer cells.

9.2.3.1 No-Threshold Models for Carcinogens

Examination of the frequency of mutations in microorganisms and experimental animals exposed to radiation or mutagenic substances has failed to demonstrate that there is a threshold dose that must be exceeded in order for genetic damage to occur. This observation has led to the general conclusion that within the limits of measurement, any dose of mutagenic chemical increases the risk of mutation. Evidence that mutations are involved in cancer (Weinberg 1983) has contributed to the general acceptance of the hypothesis that there is no threshold for substances that are initiators of carcinogenesis.

It is not possible to establish a NOEL or NOAEL for chemicals which appear to pose a finite risk at all exposure levels. Therefore, hazard evaluation for carcinogens has traditionally involved the estimation of risks at low levels of exposure. To predict low dose risk from high dose/high risk data, which is the usual situation, it is necessary to apply certain mathematical models to dose-response data. (Food Safety Council 1980; Klaassen and Doull 1980; IRLG 1979.) These models allow prediction of low dose risks based on the frequency of cancers seen at higher doses in laboratory animals or, less frequently, in humans.

For those chemicals identified as carcinogens, the unit cancer risk (UCR) is typically used as the measure of carcinogenicity. The unit risk is defined as the upper limit on the probability that the carcinogen will cause cancer at a dose of 1 mg chemical/kg body weight/day (mg/kg/d) administered over the course of a lifetime. The use of the term "upper limit" means that the actual risk is probably lower than the predicted risk and may even be zero. UCR values for the carcinogens considered in the current risk assessment are presented in Table 9-2.

The UCR values in Table 9-2 were developed using a linearized multi-stage model. It should be recognized that alternative models are available, and that these models may predict different UCR values than those shown in Table 9-2. The models available for estimating UCR values are based either on assumptions regarding the biological mechanisms that are believed to lead to cancer, or simply on the mathematical properties of particular curves. The multi-stage models appear most consistent with the biological mechanisms by which cancers are thought to be produced in an exposed population. The linearized multi-stage model, which has been accepted by numerous regulatory bodies including the United States Environmental Protection Agency, has been selected as the most appropriate basis for the development of UCR values in the current risk assessment.

9.2.3.2 Threshold Models for Carcinogens

The "no-threshold" assumption may not hold for some classes of carcinogens because they appear to act through mechanisms that involve thresholds. For example, it has been suggested by many scientists that it may be appropriate to assess the risks

TABLE 9-2

CHRONIC TOXICITY DATA USED IN THE PHASE 4B RISK ASSESSMENT
CARCINOGENS

Chemical	UCR $[(mg/kg\text{-}d)]^{-1}$	Basis	References
Organics			
Acrylonitrile	0.24	EPA unit risk based on human occupational exposure.	CAG 1985
Benzene	0.029	EPA unit risk based on human occupational exposure.	CAG 1985
Benzo(a)Anthracene	6.1 (inhal) 11.5 (oral)	ENVIRON generated based on analogy to benzo(a)pyrene.	(Analogy to Benzo(a)pyrene)
Benzo(a)Pyrene	6.1 (inhal) 11.5 (oral)	EPA based on animal oral study.	CAG 1985
Butadiene (1,3-)	1.8	EPA based on animal inhalation study.	CAG 1985
Carbon Tetrachloride	0.13	EPA based on animal oral study.	CAG 1985
Chloroform	0.081	EPA based on animal oral study.	CAG 1985
Ethylene Oxide	0.35	EPA based on animal inhalation study.	CAG 1985
Formaldehyde*	0.0112	ENVIRON calculated based on rat subchronic inhalation study.	ENVIRON 1985a
Hexachlorobenzene	1.67	EPA based on animal oral study.	CAG 1985
Hydrazine	3.9	EPA based on animal oral study.	CAG 1985
Methylene Chloride	0.014	EPA based on animal inhalation study.	CAG 1985
PCBs	4.34	EPA based on animal oral study.	CAG 1985
2,3,7,8-TCDD	1.56×10^5	EPA based on animal oral study.	CAG 1985
Tetrachloroethylene	0.051	EPA based on animal oral study.	CAG 1985
Trichloroethylene	0.011	EPA based on animal oral study.	CAG 1985
Metals			
Arsenic*	15.0	EPA based on human drinking water exposure.	CAG 1985
Beryllium	2.6 (inhal) 8.4 (oral)	EPA based on human occupational (inhalation) exposure.	CAG 1985
Cadmium*	6.1	EPA based on human occupational (inhalation) exposure.	CAG 1985
Chromium VI*	41.0	EPA based on human occupational (inhalation) exposure.	CAG 1985
Nickel*	1.05	EPA based on human occupational exposure to refinery dust.	CAG 1985

*Carcinogenic through inhalation only.

associated with exposure to chlorinated dibenzo-p-dioxins (CDD) and chlorinated dibenzofurans (CDF) using a threshold model (see, for example, OMOE 1985). Although the United States Environmental Protection Agency (USEPA) continues to assume that a threshold does not exist for CDDs and CDFs, many governments and regulatory bodies, including the Ontario Ministry of the Environment (OMOE), have concluded that a threshold dose must be exceeded for CDDs and CDFs to pose a threat to human health.

In the Phase 4A Risk Assessment, a no-threshold model was used to evaluate risks associated with exposure to all carcinogens, including CDDs and CDFs. As part of the current Phase 4B analysis, this assumption was reexamined, and it was concluded that some evidence suggests that a threshold dose for CDDs and CDFs may exist (see Appendix G). Based on the results of this review, it was decided to evaluate the risk associated with CDDs and CDFs using both threshold and no-threshold assumptions.

9.2.3.3 Toxic Equivalency of Chlorinated Dibenzo-p-Dioxins and Chlorinated Dibenzofurans

The most potent carcinogen listed in Table 9-2 is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). This chemical is generally believed to be the most toxic member of the over 200 forms of chlorinated dibenzo-p-dioxins (CDDs) and chlorinated dibenzofurans (CDFs). In the Phase 4A Generic Risk Assessment, risks associated with exposures to emissions from the proposed OWMC incinerator were driven by CDDs and CDFs, which may be formed during the combustion of hazardous wastes.

It is generally accepted that the toxicities of the various CDDs and CDFs vary considerably. In the Phase 4A risk assessment, however, it was assumed that all polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) containing four or more chlorine atoms are as toxic as 2,3,7,8-TCDD. (CDDs and CDFs with fewer than four chlorine atoms were not considered.) Since 2,3,7,8-TCDD is more potent than the only other CDDs tested for carcinogenicity to date, and is widely believed to be the most toxic of the various forms of CDD and CDF, this assumption likely overestimates the level of risk associated with exposures to mixtures of CDD and CDF.

Based on an examination of available toxicity data, a decision was made to refine the assumption that all forms of CDD and CDF are as toxic as 2,3,7,8-TCDD. Therefore, existing approaches for estimating the relative potencies of the various forms of CDD and CDF have been evaluated as part of the Phase

4B risk assessment. These approaches are based on the development of "toxic equivalency factors" for those CDDs and CDFs for which no cancer data are available.

The review of existing "toxic equivalency factor" methodologies conducted in Phase 4B is presented in Appendix H. This review indicates that schemes developed by both the Ontario Ministry of the Environment (OMOE 1985) and the United States Environmental Protection Agency (USEPA 1986b) are protective of public health. The USEPA approach, however, appears to make better use of available toxicologic data.

In the Phase 4B Risk Assessment, the risks associated with exposures to mixtures of CDD and CDF were evaluated using both threshold and no threshold models. In using the threshold model endorsed by OMOE, the "toxic equivalency factor" methodology developed by OMOE was applied to estimate the potencies of the various forms of CDD and CDF. When using the no-threshold model adopted by the USEPA, however, the USEPA "toxic equivalency factor" methodology was applied.

9.3 ACUTE TOXICITY

9.3.1 INTRODUCTION

Chemical exposures of short duration are termed acute exposures. If the acute exposure occurs to a high dose of a chemical, immediately observable effects may be produced. An acute exposure has been defined as one usually lasting 60 minutes or less, and never longer than 24 hours (NAS, 1979). A considerable body of historical data exists with respect to the risks associated with some types of acute chemical exposures (e.g., the annual risk of death from accidental exposures to drugs, pesticides, and industrial chemicals) although they are usually restricted to those situations in which a single, very high exposure has occurred. The human health effects associated with relatively low levels of exposure are less well studied.

In the risk assessment, acute exposures were evaluated using two sets of criteria: Acute Safe Exposure Limits (ASELs) and Short-Term Public Limits (STPLs). ASELs were derived from studies identifying a no-observed-effect-level (NOEL), whereas STPLs were based on the "remote possibility of a non-incapacitating, reversible effect" (NAS 1979). These two criteria are discussed separately in the following sections of the chapter.

9.3.2 ACUTE SAFE EXPOSURE LIMIT (ASEL)

The human health effects associated with acute exposures were evaluated for several chemicals considered in the risk assessment. Acute safe exposure limits (ASEL) used in these evaluations were calculated in a manner analogous to that discussed in Section 9.2.2 for developing ADI values. There are two significant differences in the approaches, however.

First, only data reported for short term exposures, as opposed to chronic exposures, were used to derive ASEL values. Second, ASEL values were based on a no-observed-effect-level (NOEL) rather than a no-observed-adverse-effect-level, (NOAEL). The short-term NOEL was divided by a safety factor developed in the same way as those used in deriving a chronic ADI (See Section 9.2.2). In most cases, human data were available for the chemicals of concern in the risk assessment, so that the application of a safety factor of 10 was deemed appropriate. In cases where a NOEL could not be identified, the lowest-observed-effect-level, (LOEL) was used to estimate the ASEL. In each case of acute exposure where a safe exposure level was estimated from a LOEL, the LOEL was based on mild effects requiring the use of either no additional safety factor or a factor of 2 in developing the ASEL.

It should be emphasized that the use of the NOEL for evaluating acute exposure is considered to be highly conservative; a NOAEL is a more commonly used basis for estimating a safe exposure level. It is generally accepted that some acute effects (such as reversible, mild irritation, odor, or sensory motor effects) are not adverse if experienced for only a short period (NRC 1979). However, to maintain consistency with the conservative assumptions adopted throughout the risk assessment, any effect (no matter how slight) was considered to be undesirable for the purpose of establishing short term safe exposure levels. A list of ASEL values developed for inhalation and oral exposures to the chemicals evaluated in the risk assessment is presented in Table 9-3.

9.3.3 SHORT TERM PUBLIC EXPOSURE LIMITS (STPLs)

Certain short-term exposures examined in the risk assessment for the proposed OWMC Facility were predicted to exceed the ASEL values discussed in Section 9.3.2. A second criterion, the STPL, was selected to evaluate these exposures.

The STPL is one of several short-term limits developed for air pollutants by the National Academy of Sciences (NAS 1979). If not exceeded, STPLs are expected to be associated with only a remote possibility of a non-capacitating, reversible effect in an exposed population. A non-incapacitating, reversible effect is defined by NAS as one that will not cause a period of

TABLE 9-3

ACUTE SAFE EXPOSURE LIMITS

Chemical	ASEL	Reference
Inhalation Exposure		
Methylene chloride	2.5 mg/kg	ENVIRON 1985a
Hydrogen chloride	2.6 mg/m^3	ENVIRON 1985a
Hydrogen cyanide	1.0 mg/m^3	ENVIRON 1985a
Hydrogen fluoride	1.3 mg/m^3	ENVIRON 1985a
Hydrogen sulfide	0.7 mg/m^3	ENVIRON 1985a
Nitrogen oxides	0.2 mg/m^3	ENVIRON 1985a
Sulfur dioxide	0.26 mg/m^3	ENVIRON 1985a
Oral Exposure		
Methylene chloride	2.5 mg/kg	ENVIRON 1985a
Petroleum distillates	4.8 mg/kg	ENVIRON 1985a
Pyridine	0.2 mg/kg	ENVIRON 1985a

Acute Safe Exposure Levels based on no-observed-effect-levels (NOELs) derived from short term studies disability; will not impair one's vision, visibility, judgment, or ability to breathe; and will not interfere with one's ability to escape from the site of the exposure.

The approach used by NAS to develop short-term limits depends on a substance-by-substance evaluation of each chemical. For those compounds for which reliable human and animal data predict little chance of irreversible effects from a single exposure, exposure limits can be established directly from the human data with no additional safety factor. If the human data are weak, but the animal data strongly suggest irreversible effects are unlikely from a single exposure, then an uncertainty (safety) factor can be applied depending on the quality of the data. When cancer or some other irreversible effects are expected, then several possible approaches can be used. These include:

- Establishing an exposure limit based on an appropriate uncertainty factor as would be done for non-carcinogens; or, depending on the nature of the chemical, include specific warnings to particular segments of the population that may be at additional risk, e.g. in the case of pregnant women exposure to a possible teratogen.
- Using quantitative risk assessment to recommend an exposure level that provides a defined level of risk (i.e. 10^{-5}, 10^{-6} etc.).

A list of STPL values developed for the risk assessment of the OWMC Facility is presented in Table 9-4.

TABLE 9-4

SHORT TERM PUBLIC LIMITS

Chemical	STPL	Reference
Inhalation Exposure		
Methylene chloride	17 mg/kg	ENVIRON 1985a
Hydrogen chloride	4 ppm for 10 min (6 mg/m^3)	ENVIRON 1985a
Sulfur dioxide	3 ppm for 10 min (7.8 mg/m^3)	ENVIRON 1985a
Oral Exposure		
Methylene chloride	17.0 mg/kg	ENVIRON 1985a
Petroleum distillates	9.7 mg/kg	ENVIRON 1985a
Pyridine	0.4 mg/kg	ENVIRON 1985a

Short-Term Public Limits based on no-observed-adverse-effect-levels (NOAELs) derived from short-term studies

9.4 ABSORPTION FACTORS

In assessing the toxicity of individual chemicals, the extent of absorption that might be expected for inhalation and ingestion exposures was examined. In order to assign an absorption factor for a specific chemical, the exposure pathway on which the ADI and/or UCR was based was identified. An absorption factor of 1.0, which corresponds to 100% absorption, was adopted for a chemical for a particular route of exposure if this route was the one in the study on which the ADI or UCR was based. If the route of exposure examined was different from the route on which the ADI or UCR was based, then absorption factors derived from the scientific literature were used in the risk assessment.

In cases where a range of the absorption factors has been reported for a particular chemical, the value representing the upper bound of the range was conservatively adopted in the risk assessment. If little or no quantitative data were available, 100 percent absorption was assumed. For example, qualitative statements regarding absorption, e.g., "readily absorbed", "rapid uptake", etc., were considered to indicate 100 percent absorption. Tables 9-5 and 9-6 list the absorption factors used in the risk assessment for oral and inhalation exposures, respectively. For those chemicals not listed in Tables 9-5 and 9-6, an absorption factor of 1.0 was assumed. An absorption factor of 0.1, which corresponds to 100% absorption, was assumed for all dermal exposures.

TABLE 9-5

SUMMARY OF ORAL ABSORPTION DATA

Chemical	Absorption Factor[1]	Reference
Organic Compounds		
Acrylonitrile	0.95	Young *et al*. 1977, in USEPA 1983
Benzene	0.5	Health and Welfare Canada 1979
Dioxins/Furans	0.25[2] 1.00[3]	Derived from Van den Berg *et al*. 1983
Metals		
Cadmium	0.1	NRC 1977
Chromium VI	0.5	USEPA 1984c
Copper	0.4	USEPA 1980b

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[1] The absorption factor represents the fraction of the chemical which is absorbed; thus an absorption factor of 1.0 corresponds to 100% absorption, a factor of 0.1 corresponds to 10% absorption.

[2] If bound to particulates.

[3] If not bound to particulates.

TABLE 9-6

SUMMARY OF INHALATION ABSORPTION DATA

Chemical	Absorption Factor[1]	Reference
Organic Compounds		
Chloroform	0.7	USEPA 1980c
Phenol	0.8	Piotrowski 1971 in USEPA 1980g
Styrene	0.66	WHO 1983
Tetrachloroethylene	0.57	Ogata *et al*. 1971 in USEPA 1980d
Tetraethyl lead	0.4	USEPA 1977
Trichloroethylene	0.7	Soucek and Ulachova 1960, in NIOSH 1973
Metals		
Arsenic	0.85	USEPA 1980e
Lead	0.46	USEPA 1984d
Mercury	0.80	USEPA 1980f

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[1] The absorption factor represents the fraction of the chemical which is absorbed; thus an absorption factor of 1.0 corresponds to 100% absorption, a factor of 0.1 corresponds to 10% absorption.

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CHAPTER 10

PUBLIC HEALTH RISKS ASSOCIATED WITH THE OPERATION OF THE OWMC FACILITY

10.1 INTRODUCTION

Potentially hazardous chemicals will be released to the environment as a result of the operation of the proposed OWMC Facility. Using the information derived from the examination of the nature and magnitude of potential human exposures (Chapters 5, 7, and 8) and the toxicity of the chemicals of concern (Chapter 9), quantitative estimates of the public health risks associated with the operation of the OWMC Facility were developed in the risk assessment.

In this chapter, the general methodology used to characterize human health risks is described, and the predicted risk values are presented. Health risks have been characterized separately for (1) long-term (chronic) exposure to those chemicals that have the potential to cause cancer, i.e., carcinogens; (2) chronic exposure to those chemicals not believed to have the potential to cause cancer, i.e., non-carcinogens; and (3) short-term (acute) exposures.

The risk estimates reported in this chapter correspond to the operation of a 150 000 t/a facility. A sensitivity analysis has also been conducted to examine the potential risks associated with a 300 000 t/a facility. The results of that analysis are presented in Appendix N.

10.2 CHRONIC EXPOSURE TO CARCINOGENS

10.2.1 METHODOLOGY

For chronic exposure to carcinogens, risk is typically characterized as the incremental probability of an individual's contracting cancer during his lifetime, and can be calculated by multiplying the risk per unit dose (UCR) by the estimated lifetime daily dose:

$$\text{Cancer Risk} = (\text{UCR})\ (\text{DHD})$$

Where:

Cancer Risk = Cancer Risk for exposure to a given chemical

UCR = Unit Cancer Risk $[\mathrm{mg/(kg\text{-}d)}]^{-1}$

DHD = Daily Human Dose, mg/(kg-d)

It should be noted that the value calculated using this equation corresponds to the incidence of cancer, and is not equivalent to the risk of dying from cancer.

It has been assumed in the current analysis that the risks associated with exposures to individual carcinogens can be added to provide an estimate of total cancer risk. It is recognized that this assumption can lead to an overestimate or underestimate of risk if antagonistic or synergistic (respectively) interactions occur. In general, however, a thorough understanding of the mechanisms of how specific mixtures of chemicals will interact is not available. Although some studies have been conducted involving two component mixtures, they have limited application assessing interactions in a mixture containing three or more chemicals. For example, if two compounds interact (either synergistically or antagonistically) the inclusion of additional substances could substantially alter the nature and degree of toxicologic interaction.

The preferred approach for assessing risk associated with exposure to chemical mixtures is to use toxicologic data specific to the mixture of concern. In practice, however, little or no quantitative information is available on the interactions within most chemical mixtures and, therefore, an alternative procedure must be adopted to assess total risk. While in Canada a procedure for evaluating the total cancer risk associated with exposures to complex chemical mixtures has not been widely accepted, the United States Environmental Protection Agency has recommended an additive model (USEPA 1985). This approach has been used in the risk assessment.

The toxic effect caused by exposure to a chemical can be more or less severe in a child than in an adult due to variations in body weights, metabolic rates, and other factors. In most cases, a child less than 10 to 12 years of age will receive somewhat higher doses per kilogram of body weight than will an adult. For 70-year chronic exposures, however, the fraction of time an individual is a child is small relative to the period of exposure. Furthermore, toxicological investigations for chronic exposures to carcinogens are conducted essentially over the entire life of the laboratory animal (after weaning until death) and therefore take into account possible changes in metabolism that occur over time. For these reasons, chronic exposures to carcinogens were generally evaluated in the risk assessment by examining the dose predicted to be received by an adult. The risks associated with dermal contact with, and ingestion of, soil were calculated separately for adults and children, however, since children may exhibit significantly different patterns of exposure through these pathways.

10.2.2 ESTIMATED RISKS

10.2.2.1 Incinerator Emissions

The risks associated with exposure to incinerator emissions were determined using two separate sets of assumptions regarding the toxicity of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF):

- PCDD/PCDF were considered to be no-threshold carcinogens, and the United States Environmental Protection Agency (USEPA) toxic equivalency scheme was applied to estimate the toxicity of the various forms of PCDD/PCDF; and
- PCDD/PCDF were treated as threshold carcinogens, and the Ontario Ministry of the Environment (OMOE) toxic equivalency scheme for these compounds was applied.

Assumptions with respect to the toxicity of chemicals of concern other than PCDD/PCDF remained the same in the two cases examined. The estimated lifetime cancer risks based on the different assumptions regarding PCDD/PCDF toxicity are presented in Tables 10-1 and 10-2. Risk estimates for each human exposure pathway evaluated are reported separately.

A review of Tables 10-1 and 10-2 shows that none of the pathways examined pose a risk exceeding 8×10^{-7}, or a probability of eight-in-ten-million of developing cancer, and that the pathways contributing most significantly to total risk are direct inhalation and ingestion of meat and milk. As indicated on Table 10-1, consumption of meat and milk contribute more heavily to total risk if it is assumed that PCDD and PCDF act as no-threshold carcinogens. The total risk from all pathways combined is approximately 2×10^{-6}, or two-in-one-million, if PCDD/PCDF are considered to be no-threshold carcinogens.

TABLE 10-1

CONSERVATIVE ESTIMATE OF LIFETIME CANCER RISK: EXPOSURE TO INCINERATOR EMISSIONS

CASE 1: PCDD/PCDF ASSUMED TO BE NO-THRESHOLD CARCINOGENS[1]

(150 000 t/a Facility)

Exposure Pathway	Lifetime Cancer Risks[3]
Inhalation[2]	2×10^{-7}
Beef	8×10^{-7}
Milk	3×10^{-7}
Pork	1×10^{-7}
Poultry	2×10^{-7}
Eggs	8×10^{-8}
Vegetables and Fruit	7×10^{-8}
Soil Ingestion	8×10^{-10}
Soil Contact	5×10^{-9}
Pond	3×10^{-8}
Cistern	4×10^{-8}

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[1] Polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) are assumed to be no-threshold carcinogens. The toxicity of the PCDD/PCDF mixture has been estimated using the USEPA toxic equivalency scheme.

[2] Based on the assumption that 10% of the chromium emitted is in the hexavalent form (Cr VI).

[3] Expressed as the probability of developing cancer. Thus, if the lifetime cancer risk is 1×10^{-6}, an exposed individual has a one in one million chance of developing cancer; at a risk level of 1×10^{-7}, the individual has a one in ten million chance of developing cancer.

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-2

CONSERVATIVE ESTIMATE OF LIFETIME CANCER RISK: EXPOSURE TO INCINERATOR EMISSIONS
CASE 2: PCDD/PCDF ASSUMED TO BE THRESHOLD CARCINOGENS[1]
(150 000 t/a Facility)

Exposure Pathway	Lifetime Cancer Risks[3]
Inhalation[2]	2×10^{-7}
Beef	2×10^{-7}
Milk	3×10^{-7}
Pork	5×10^{-8}
Poultry	7×10^{-8}
Eggs	4×10^{-8}
Vegetables and Fruit	5×10^{-8}
Soil Ingestion	6×10^{-10}
Soil Contact	2×10^{-9}
Pond	3×10^{-8}
Cistern	1×10^{-8}

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[1] Polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) are assumed to be threshold carcinogens. The toxicity of the PCDD/PCDF mixture has been estimated using the OMOE toxic equivalency scheme.

[2] Based on the assumption that 10% of the chromium emitted is in the hexavalent form (Cr VI).

[3] Expressed as the possibility of developing cancer. Thus, if the lifetime cancer risk is 1×10^{-6}, an exposed individual has a one in one million chance of developing cancer; at a risk level of 1×10^{-7}, the individual has a one in ten million chance of developing cancer.

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

The estimates of cancer risk associated with exposure to the individual constituents of the incinerator emissions are presented in Tables 10-3 through 10-6. As shown in Table 10-3, chromium contributes significantly to the risks associated with inhalation exposure. The estimated magnitude of the cancer risk for chromium is based on the assumption that 10 percent of the chromium emitted from the incinerator stack is in the hexavalent form.

Chromium compounds can exist in any of three oxidation states: elemental (Cr), trivalent (Cr III), and hexavalent (Cr VI). The toxicity of chromium varies according to the oxidation state. Cr VI is the most hazardous form of chromium; it is generally considered to be one of the most potent human carcinogens via inhalation exposure. In contrast, Cr and Cr III have not been shown to be carcinogenic in either humans or laboratory animals.

As discussed in detail in Appendix C, chromium emitted from combustion sources is believed to be primarily a mixture of the trivalent and hexavalent forms. Although information on the distribution between the two oxidation states in incinerator emissions is very limited, it is generally believed that trivalent chromium predominates (see, for example, CARB 1985). For example, the incinerator technology consultants to OWMC, Von Roll, have estimated that only approximately

TABLE 10-3

CANCER RISKS ASSOCIATED WITH EXPOSURE TO INCINERATOR EMISSIONS VIA INHALATION

CHEMICAL CONSTITUENT	INHALATION CANCER RISK
ACETONE	0.00E+00
ACRYLONITRILE	3.89E-10
BENZENE	1.58E-09
BENZO(A)ANTHRACENE	0.00E+00
BENZO(A)PYRENE	0.00E+00
BROMODICHLOROMETHANE	1.13E-08
BROMOFORM	8.71E-10
BUTADIENE,1,3-	2.60E-10
CARBON TETRACHLORIDE	1.07E-09
CHLOROBENZENE	0.00E+00
CHLOROFORM	4.83E-09
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.02E-08
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	4.32E-11
FORMALDEHYDE	4.89E-11
HEXACHLOROBENZENE	1.16E-10
HYDRAZINE	1.58E-12
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	1.91E-09
NAPHTHALENE	0.00E+00
NITROPHENOL,o-	0.00E+00
PCB	7.58E-10
PETROLEUM DISTILLATES	0.00E+00
PHENOL	0.00E+00
PYRIDINE	0.00E+00
STYRENE	0.00E+00
TETRACHLOROETHYLENE	5.94E-09
TETRAETHYL LEAD	0.00E+00
TOLUENE	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00
TRICHLOROETHYLENE	1.40E-09
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	0.00E+00
ARSENIC	8.03E-09
BERYLLIUM	1.11E-11
CADMIUM	5.79E-08
CHROMIUM (VI)	9.23E-08
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	6.70E-09
ZINC	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-4

CANCER RISKS ASSOCIATED WITH EXPOSURE TO INCINERATOR EMISSIONS VIA INGESTION OF PRODUCE, MEAT, AND ANIMAL PRODUCTS

CHEMICAL CONSTITUENT	PRODUCE CANCER RISK	BEEF CANCER RISK	MILK CANCER RISK	PORK CANCER RISK	POULTRY CANCER RISK	EGG CANCER RISK
ACETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ACRYLONITRILE	1.16E-07	1.24E-15	5.99E-15	2.10E-13	1.51E-13	8.21E-14
BENZENE	1.04E-10	1.16E-14	4.52E-14	2.81E-13	2.26E-13	1.22E-13
BENZO(A)ANTHRACENE	2.53E-08	2.77E-09	4.65E-08	1.71E-08	3.55E-08	1.94E-08
BENZO(A)PYRENE	1.05E-08	3.23E-08	5.51E-08	1.32E-08	2.49E-08	1.56E-08
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	6.89E-12	8.37E-16	2.49E-15	1.68E-14	3.15E-16	1.72E-14
CARBON TETRACHLORIDE	7.38E-11	5.65E-14	1.85E-13	5.60E-13	5.21E-13	2.84E-13
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	1.22E-09	8.82E-14	3.59E-13	2.61E-12	1.94E-12	1.06E-12
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.35E-08	6.00E-07	2.00E-07	4.57E-08	7.99E-08	4.35E-08
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
FORMALDEHYDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
HEXACHLOROBENZENE	1.33E-09	1.07E-07	3.53E-08	1.65E-08	5.89E-09	3.21E-09
HYDRAZINE	3.94E-11	9.83E-19	1.21E-18	7.06E-16	6.67E-16	3.64E-16
METHYL ETHYL KETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	6.03E-10	5.10E-15	6.00E-15	3.12E-13	2.50E-13	1.36E-13
NAPHTHALENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	2.68E-09	4.65E-08	5.94E-09	2.75E-09	3.66E-09	1.99E-09
PETROLEUM DISTILLATES	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	1.37E-09	5.15E-13	5.27E-13	7.29E-12	5.37E-12	2.93E-12
TETRAETHYL LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TRICHLOROETHYLENE	2.06E-10	4.99E-14	5.29E-14	8.52E-13	7.09E-13	3.86E-13
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BERYLLIUM	1.11E-09	1.39E-10	6.12E-13	1.63E-11	6.31E-13	3.00E-13
CADMIUM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHROMIUM (VI)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
COPPER	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-5

CANCER RISKS ASSOCIATED WITH EXPOSURE TO INCINERATOR EMISSIONS VIA SOIL INGESTION AND DERMAL CONTACT WITH SOIL

CHEMICAL CONSTITUENT	SOIL INGESTION RISK	SOIL CONTACT RISK
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	3.88E-13	1.39E-12
BENZENE	7.18E-13	2.58E-12
BENZO(A)ANTHRACENE	2.71E-10	9.74E-10
BENZO(A)PYRENE	1.29E-10	4.65E-10
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.00E-14	7.17E-14
CARBON TETRACHLORIDE	6.23E-13	2.24E-12
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	3.44E-12	1.24E-11
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	2.20E-10	3.15E-09
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	5.05E-14	1.81E-13
FORMALDEHYDE	0.00E+00	0.00E+00
HEXACHLOROBENZENE	2.92E-11	1.05E-10
HYDRAZINE	1.59E-15	5.71E-15
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	4.61E-13	1.66E-12
NAPHTHALENE	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	9.49E-11	3.41E-10
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	8.62E-12	3.09E-11
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00
TRICHLOROETHYLENE	1.03E-12	3.70E-12
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00
BERYLLIUM	1.95E-11	7.00E-11
CADMIUM	0.00E+00	0.00E+00
CHROMIUM (VI)	0.00E+00	0.00E+00
COPPER	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-6

CANCER RISKS ASSOCIATED WITH EXPOSURE TO INCINERATOR EMISSIONS VIA INGESTION OF POND AND CISTERN WATER

CONTAMINANT	CISTERN CANCER RISK	POND CANCER RISK
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	8.93E-12	4.56E-11
BENZENE	3.05E-13	3.74E-11
BENZO(A)ANTHRACENE	2.91E-09	1.09E-08
BENZO(A)PYRENE	2.40E-09	1.53E-08
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.67E-15	1.21E-11
CARBON TETRACHLORIDE	7.46E-14	5.06E-11
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	4.00E-12	3.27E-10
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	2.96E-09	4.77E-09
DIOXIN,2,3,7,8-EQ(MCE)	0.00E+00	0.00E+00
ETHANOL	[illegible]	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00
FORMALDEHYDE	0.00E+00	0.00E+00
HEXACHLOROBENZENE	3.63E-13	1.05E-11
HYDRAZINE	2.54E-10	7.32E-14
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	1.86E-12	1.02E-10
NAPHTHALENE	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	6.09E-13	2.90E-11
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	1.25E-12	4.94E-10
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00
TRICHLOROETHYLENE	4.43E-13	9.47E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00
BERYLLIUM	6.85E-09	6.44E-10
CADMIUM	0.00E+00	0.00E+00
CHROMIUM (VI)	0.00E+00	0.00E+00
COPPER	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

1 percent of the total chromium emissions from the facility will be in the hexavalent form (Environmental Elements 1986).

Taken as a whole, available information indicates that it is unlikely that greater than 10 percent of the chromium emissions from the OWMC incinerator will be Cr VI. If it is assumed, however, that 100 percent of the chromium emissions are hexavalent, the predicted cancer risk associated with inhalation exposure would increase:

Chromium Emissions from the Incinerator Stack	Lifetime Cancer Risk, Inhalation of Incinerator Emissions
100% Cr VI	1×10^{-6}
10% Cr VI	2×10^{-7}

If, on the other hand, the 1 percent Cr VI estimate developed by the incinerator technology consultants is assumed, chromium emissions would not have a significant impact on total lifetime cancer risk.

Table 10-4 suggests that the risks associated with the food pathways (beef, milk products, pork, poultry, eggs, and vegetables/fruit) are driven primarily by PCDD, PCDF, polychlorinated biphenyls (PCBs) and other persistent organic chemicals. In interpreting these risks, it should be recognized that the following highly conservative assumptions have been made:

- All beef, milk products, pork, poultry and eggs consumed by an individual over an entire lifetime are taken exclusively from the area of maximum potential contamination. Fodder and water ingested by the livestock is also assumed to come only from this location.
- All fruits and vegetables (except those which could not be grown in the site vicinity) consumed by an individual over an entire lifetime come exclusively from the area of maximum potential contamination.

Elements of these assumptions, taken individually, are highly conservative but not entirely implausible. For example, it is possible that a farm may be located in the area of maximum potential impact. It is, however, unlikely that the entire 70-year food supply for an individual would be taken from this farm. A more likely scenario would involve a diet consisting of food both produced in the region and imported from other areas.

It should be noted that there are significant uncertainties associated with the prediction of chemical exposures through the consumption of food. Methods for estimating the extent of chemical uptake by plants are limited. Furthermore, limited information on the bioaccumulation of specific chemicals, such as 2,3,7,8 tetrachlorinated dibenzo-p-dioxin (2,3,7,8-TCDD), in certain foods, such as milk, are available. The extent of bioaccumulation of most contaminants in most foods, however, were indirectly estimated using available data on the physical properties of the chemicals of concern (see Appendix D).

As shown in Tables 10-5 and 10-6, the risks associated with dermal contact with soil and with the ingestion of pond water, cistern water, and soil are significantly lower than those predicted for the inhalation or food consumption pathways. A conservative estimate of the lifetime cancer risk associated with each of these pathways is approximately one-in-ten-million, or 1×10^{-7}, or less.

10.2.2.2 Evaporator Emissions

The incremental cancer risks associated with evaporator emissions (which include a consideration of releases from the make-up water sent to the incinerator scrubbing system) are presented in Table 10-7. No pathway poses an incremental cancer risk exceeding 1×10^{-7}, or a probability of one-in-ten-million of developing cancer. The cancer risks estimated for exposures to chemicals released from the evaporator are lower than those predicted for incinerator emissions for all exposure pathways examined.

The estimated lifetime cancer risks associated with exposure to individual chemicals in the evaporator emissions are presented in Tables 10-8 through 10-11. As shown on these tables, the cancer risks are driven primarily by volatile organic chemicals (such as acrylonitrile) for the inhalation pathway and polyaromatic hydrocarbons (PAH), such as benzo(a)pyrene, for the other exposure pathways.

TABLE 10-7

CONSERVATIVE ESTIMATE OF LIFETIME CANCER RISK: EXPOSURE TO EVAPORATOR EMISSIONS

(150,000 t/a Facility)

Exposure Pathway	Lifetime Cancer Risks[1]
Inhalation	8×10^{-9}
Beef	9×10^{-9}
Milk	1×10^{-8}
Pork	4×10^{-9}
Poultry	7×10^{-9}
Eggs	4×10^{-9}
Vegetables and Fruit	2×10^{-8}
Soil Ingestion	4×10^{-11}
Soil Contact	1×10^{-10}
Pond	5×10^{-9}
Cistern	8×10^{-10}

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[1] Expressed as the probability of developing cancer. Thus, if the lifetime cancer risk is 1×10^{-6}, an exposed individual has a one in one million chance of developing cancer; at a risk level of 1×10^{-7}, the individual has a one-in-ten-million chance of developing cancer.

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-8

CANCER RISKS ASSOCIATED WITH EXPOSURE TO EVAPORATOR EMISSIONS VIA INHALATION

CHEMICAL CONSTITUENT	INHALATION CANCER RISK
ACETONE	0.00E+00
ACRYLONITRILE	5.19E-09
BENZENE	1.21E-10
BENZO(A)ANTHRACENE	0.00E+00
BENZO(A)PYRENE	0.00E+00
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.89E-09
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	1.12E-12
HEXACHLOROBENZENE	0.00E+00
HYDRAZINE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	0.00E+00
NITROPHENOL,o-	0.00E+00
PCB	1.81E-11
PETROLEUM DISTILLATES	0.00E+00
PHENOL	0.00E+00
PYRIDINE	0.00E+00
STYRENE	0.00E+00
TETRACHLOROETHYLENE	4.09E-10
TETRAETHYL LEAD	0.00E+00
TOLUENE	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00
TRICHLOROETHYLENE	5.47E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	0.00E+00
ARSENIC	6.50E-12
BERYLLIUM	0.00E+00
CADMIUM	6.22E-14
CHROMIUM (VI)	2.09E-12
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	5.35E-13
ZINC	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-9

CANCER RISKS ASSOCIATED WITH EXPOSURE TO EVAPORATOR EMISSIONS VIA INGESTION OF PRODUCE, MEAT, AND ANIMAL PRODUCTS

CHEMICAL CONSTITUENT	PRODUCE CANCER RISK	BEEF CANCER RISK	MILK CANCER RISK	PORK CANCER RISK	POULTRY CANCER RISK	EGG CANCER RISK
ACETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ACRYLONITRILE	1.63E-08	1.74E-14	8.41E-14	2.95E-12	2.11E-12	1.15E-12
BENZENE	1.59E-11	1.77E-15	6.92E-15	4.30E-14	3.39E-14	1.85E-14
BENZO(A)ANTHRACENE	8.27E-10	9.00E-10	1.51E-09	5.55E-10	1.16E-09	6.31E-10
BENZO(A)PYRENE	2.41E-09	7.39E-09	1.26E-08	3.03E-09	5.71E-09	3.12E-09
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	5.02E-11	6.07E-15	1.81E-14	1.22E-13	2.28E-13	1.24E-13
CARBON TETRACHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
FORMALDEHYDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
HEXACHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	6.40E-11	1.00E-09	5.05E-10	6.28E-11	8.38E-11	4.57E-11
PETROLEUM DISTILLATES	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	9.41E-11	3.51E-14	1.28E-13	4.99E-13	3.62E-13	1.98E-13
TETRAETHYL LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TRICHLOROETHYLENE	8.05E-12	1.94E-15	7.13E-15	3.32E-14	2.74E-14	1.49E-14
TRICHLOROTRIFLUOROETHAN	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHROMIUM (VI)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
COPPER	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-10

CANCER RISKS ASSOCIATED WITH EXPOSURE TO EVAPORATOR EMISSIONS VIA SOIL INGESTION AND DERMAL CONTACT WITH SOIL

CHEMICAL CONSTITUENT	SOIL INGESTION RISK	SOIL CONTACT RISK
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	4.39E-12	1.50E-11
BENZENE	4.67E-14	1.59E-13
BENZO(A)ANTHRACENE	7.52E-12	2.56E-11
BENZO(A)PYRENE	2.53E-11	8.61E-11
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	1.23E-13	4.20E-13
CARBON TETRACHLORIDE	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00
FORMALDEHYDE	0.00E+00	0.00E+00
HEXACHLOROBENZENE	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	1.92E-12	6.54E-12
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	5.03E-13	1.71E-12
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00
TRICHLOROETHYLENE	3.42E-14	1.17E-13
TRICHLOROTRIFLUOROETHAN	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00
CHROMIUM (VI)	0.00E+00	0.00E+00
COPPER	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-11

CANCER RISKS ASSOCIATED WITH EXPOSURE TO EVAPORATOR EMISSIONS VIA INGESTION OF POND AND CISTERN WATER

CONTAMINANT	CISTERN CANCER RISK	POND CANCER RISK
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	1.25E-10	6.40E-10
BENZENE	4.67E-14	5.74E-12
BENZO(A)ANTHRACENE	9.62E-11	3.56E-10
BENZO(A)PYRENE	5.52E-10	3.52E-09
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	1.94E-14	8.79E-11
CARBON TETRACHLORIDE	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00
FORMALDEHYDE	0.00E+00	0.00E+00
HEXACHLOROBENZENE	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00
NAPHTHALENE	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	1.45E-14	6.93E-13
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	8.62E-14	3.40E-11
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00
TRICHLOROETHYLENE	1.73E-14	3.71E-12
TRICHLOROTRIFLUOROETHAN	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00
ARSENIC	0.00E+00	0.00E+00
BERYLLIUM	0.00E+00	0.00E+00
CADMIUM	0.00E+00	0.00E+00
CHROMIUM (VI)	0.00E+00	0.00E+00
COPPER	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00
NICKEL	0.00E+00	0.00E+00
ZINC	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

Due to the scarcity of data with which to predict evaporator emissions, a separate analysis was conducted by the engineering consultants to OWMC (Monenco 1987). This analysis relied on wastewater data compiled by the United States Environmental Protection Agency (USEPA 1983) as well as information developed for OWMC by CANVIRO Consultants (1985) to predict potential releases from the evaporator stack. The results of the assessment, which are included in Appendix M, indicate that inhalation exposure to the contaminants evaluated will pose an incremental cancer risk of significantly less than one-in-one-million, i.e., 1×10^{-6}.

10.2.2.3 Fugitive Emissions

The estimated lifetime cancer risks associated with exposure to fugitive emissions are reported in Table 10-12 for each of the pathways examined. As shown in the table, the most significant pathways for exposure are predicted to be direct inhalation and consumption of contaminated produce. Other potential exposure pathways are expected to pose much lower risks since fugitive releases will consist primarily of volatile organic chemicals that are not likely to accumulate significantly in the environment.

In Table 10-12, a risk of approximately one-in-one-million, or 1×10^{-6}, is estimated for inhalation exposure to fugitive

TABLE 10-12

CONSERVATIVE ESTIMATE OF LIFETIME CANCER RISK: EXPOSURE TO FUGITIVE EMISSIONS

(150,000 t/a Facility)

Exposure Pathway	Lifetime Cancer Risks[1]
Inhalation	9×10^{-7}
Beef	5×10^{-9}
Milk	2×10^{-9}
Pork	5×10^{-10}
Poultry	5×10^{-10}
Eggs	3×10^{-10}
Vegetables and Fruit	2×10^{-7}
Soil Ingestion	3×10^{-10}
Soil Contact	1×10^{-9}
Pond	5×10^{-8}
Cistern	6×10^{-9}

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[1] Expressed as the probability of developing cancer. Thus, if the lifetime cancer risk is 1×10^{-6}, an exposed individual has a one in one million chance of developing cancer; at a risk level of 1×10^{-7}, the individual has a one-in-ten-million chance of developing cancer.

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

emissions. This level of risk corresponds to a 70-year continuous exposure occurring in the area of maximum off-site concentrations. It is unlikely that actual risks will approach this level since it was assumed that the exposed individual will be located near the boundary of the site 24 hours a day for an entire lifetime.

The second most significant pathway for exposure to fugitive emissions was shown to be the ingestion of crops grown near the preferred site. It should be recognized, however, that the following assumptions were made in calculating chemical concentrations in plants:

- Volatile chemicals which deposit onto the soil will instantaneously mix to a depth of 15 cm. In most cases, volatile chemicals would not be expected to mix to 15 cm, but would, instead, volatilize from the top layer of the soil to the atmosphere.
- Biodegradation of chemicals in soil has not been considered. Losses due to biodegradation may be significant for certain volatile organics, such as formaldehyde.
- All crops consumed over an entire lifetime will be grown near the facility boundary.

The lifetime cancer risks for the individual chemicals present in fugitive emissions are shown on Tables 10-13 through 10-16. The chemicals contributing significantly to the incremental risks are volatile organics such as methylene chloride and tetrachloroethylene.

TABLE 10-13

CANCER RISKS ASSOCIATED WITH EXPOSURE TO FUGITIVE EMISSIONS VIA INHALATION

CHEMICAL CONSTITUENT	INHALATION CANCER RISK
ACETONE	0.00E+00
ACRYLONITRILE	3.48E-08
BENZENE	1.65E-08
BENZO(A)ANTHRACENE	0.00E+00
BENZO(A)PYRENE	0.00E+00
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	3.98E-07
CARBON TETRACHLORIDE	3.40E-08
CHLOROBENZENE	0.00E+00
CHLOROFORM	3.69E-08
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	8.96E-10
FORMALDEHYDE	8.21E-08
HEXACHLOROBENZENE	1.55E-15
HYDRAZINE	3.07E-11
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	1.85E-07
NAPHTHALENE	0.00E+00
NITROPHENOL,o-	0.00E+00
PCB	7.59E-11
PETROLEUM DISTILLATES	0.00E+00
PHENOL	0.00E+00
PYRIDINE	0.00E+00
STYRENE	0.00E+00
TETRACHLOROETHYLENE	1.05E-07
TETRAETHYL LEAD	0.00E+00
TOLUENE	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00
TRICHLOROETHYLENE	3.76E-08
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-14

CANCER RISKS ASSOCIATED WITH EXPOSURE TO FUGITIVE EMISSIONS VIA INGESTION OF PRODUCE, MEAT, AND ANIMAL PRODUCTS

CHEMICAL CONSTITUENT	PRODUCE CANCER RISK	BEEF CANCER RISK	MILK CANCER RISK	PORK CANCER RISK	POULTRY CANCER RISK	EGG CANCER RISK
ACETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ACRYLONITRILE	1.09E-07	1.16E-13	5.63E-13	1.97E-11	1.41E-11	7.70E-12
BENZENE	2.16E-09	2.41E-13	9.42E-13	5.85E-12	4.62E-12	2.52E-12
BENZO(A)ANTHRACENE	1.32E-16	1.44E-16	2.41E-16	8.85E-17	1.85E-16	1.01E-16
BENZO(A)PYRENE	5.45E-17	1.67E-16	2.85E-16	6.84E-17	1.29E-16	7.04E-17
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	1.06E-08	1.28E-12	3.81E-12	2.57E-11	4.80E-11	2.62E-11
CARBON TETRACHLORIDE	2.35E-09	1.78E-12	5.85E-12	1.77E-11	1.63E-11	8.89E-12
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	9.29E-09	6.70E-13	2.73E-12	1.99E-11	1.46E-11	7.99E-12
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE OXIDE	6.68E-09	1.96E-15	9.60E-15	5.48E-13	4.28E-13	2.33E-13
FORMALDEHYDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
HEXACHLOROBENZENE	1.77E-13	1.42E-12	1.86E-12	2.20E-13	7.78E-14	4.24E-14
HYDRAZINE	7.70E-10	1.91E-17	9.42E-17	1.37E-14	1.30E-14	7.08E-15
METHYL ETHYL KETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	3.88E-08	4.90E-13	2.14E-12	3.01E-11	2.40E-11	1.31E-11
NAPHTHALENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NITROPHENOL,O-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	2.69E-10	4.50E-09	2.11E-09	2.62E-10	3.50E-10	1.91E-10
PETROLEUM DISTILLATES	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	2.43E-08	9.05E-12	3.31E-11	1.29E-10	9.34E-11	5.09E-11
TETRAETHYL LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TRICHLOROETHYLENE	5.54E-09	1.33E-12	4.91E-12	2.28E-11	1.88E-11	1.03E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-15

CANCER RISKS ASSOCIATED WITH EXPOSURE TO FUGITIVE EMISSIONS VIA SOIL INGESTION AND DERMAL CONTACT WITH SOIL

CHEMICAL CONSTITUENT	SOIL INGESTION RISK	SOIL CONTACT RISK
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	2.94E-11	1.00E-10
BENZENE	6.36E-12	2.17E-11
BENZO(A)ANTHRACENE	1.20E-18	4.09E-18
BENZO(A)PYRENE	5.72E-19	1.95E-18
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.60E-11	8.85E-11
CARBON TETRACHLORIDE	1.68E-11	5.74E-11
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	2.23E-11	7.59E-11
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	8.88E-13	3.03E-12
FORMALDEHYDE	0.00E+00	0.00E+00
HEXACHLOROBENZENE	3.30E-16	1.12E-15
HYDRAZINE	2.62E-14	8.95E-14
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	3.77E-11	1.29E-10
NAPHTHALENE	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	8.05E-12	2.75E-11
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	1.30E-10	4.43E-10
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00
TRICHLOROETHYLENE	2.35E-11	8.03E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-16

CANCER RISKS ASSOCIATED WITH EXPOSURE TO FUGITIVE EMISSIONS VIA INGESTION OF POND AND CISTERN WATER

CONTAMINANT	CISTERN CANCER RISK	POND CANCER RISK
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	8.39E-10	4.29E-09
BENZENE	6.36E-12	7.81E-10
BENZO(A)ANTHRACENE	1.54E-17	5.68E-17
BENZO(A)PYRENE	1.25E-17	7.96E-17
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	4.09E-12	1.85E-08
CARBON TETRACHLORIDE	2.38E-12	1.61E-09
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	3.05E-11	2.50E-09
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	5.25E-11	1.10E-10
FORMALDEHYDE	0.00E+00	0.00E+00
HEXACHLOROBENZENE	4.81E-18	1.39E-16
HYDRAZINE	4.94E-09	1.42E-12
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	1.79E-10	9.79E-09
NAPHTHALENE	0.00E+00	0.00E+00
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	6.17E-14	2.91E-12
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	0.00E+00	0.00E+00
PYRIDINE	0.00E+00	0.00E+00
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	2.23E-11	8.78E-09
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	0.00E+00	0.00E+00
TRICHLOROETHANE,1,1,1-	0.00E+00	0.00E+00
TRICHLOROETHYLENE	1.19E-11	2.55E-09
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00
XYLENE	0.00E+00	0.00E+00

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

10.3 CHRONIC EXPOSURE TO NON-CARCINOGENS

10.3.1 METHODOLOGY

As discussed in Chapter 9, the risks associated with chronic exposures to non-carcinogens are typically characterized differently from those for exposures to carcinogens. For non-carcinogens, the predicted lifetime daily dose of the chemical is compared to the Acceptable Daily Intake (ADI) value to calculate an Adjusted Margin of Safety (AMOS):

$$AMOS = \frac{ADI}{DHD}$$

Where:

AMOS = Adjusted Margin of Safety, unitless

ADI = Acceptable Daily Intake, mg/(kg-d)

DHD = Daily Human Dose, mg/(kg-d)

AMOS values calculated using this formula correspond to exposures to individual chemicals. As discussed in Section 10.2.1, a thorough understanding of how specific mixtures of chemicals will interact, and thus influence the level of total risk, is not available. In Canada, a methodology for evaluating the risk associated with exposures to mixtures of chemicals has not been widely accepted. The United States Environmental Protection Agency, however, has proposed guidelines for the assessment of health risks associated with exposures to mixtures of non-carcinogens (USEPA 1985). An aggregation methodology based on these guidelines has been used in the risk assessment.

In evaluating the total risk associated with exposures to mixtures of non-carcinogens, AMOS values were aggregated for each organ or system of the body using the following formula:

$$\text{Aggregated AMOS} = [\sum (1/\text{AMOS})]^{-1}$$

Aggregation of individual AMOS values using this equation allows for an estimation of the daily burden placed on an organ due to exposure to a mixture of non-carcinogenic substances. The methodology is conservative, however, since the toxic mechanisms of many chemicals may be independent.

In assessing the results of the analysis of chronic exposures to non-carcinogens, an aggregated AMOS value equal to or greater than one was considered to represent a situation in which the risk to public health is very small. In such cases, the predicted level of exposure is below that considered to be sufficient to cause an adverse health effect. An aggregated AMOS less than one indicates that the predicted exposure may be significant, and could lead to adverse human health effects.

The derivation of an ADI value includes the application of a safety factor to account for the presence of sensitive individuals, such as children, in the exposed population. In most cases, however, the dose received by a young child will be somewhat larger per kilogram body weight than the dose received by an adult. For example, a ten-year-old weighing 31 kg with a breathing rate of 12 m^3/d would receive a dose approximately 15-20 percent greater than would a 70 kg adult with a breathing rate of 23 m^3/d. Therefore, exposures to non-carcinogens were evaluated for children as well as adults, although the base case considered the dose received by an adult.

10.3.2 ESTIMATED RISKS

Aggregated AMOS values corresponding to incinerator, evaporator, and fugitive emissions are shown in Tables 10-17, 10-18, and 10-19, respectively. The aggregated AMOS values for each exposure pathway examined are presented separately for each organ or system of the body. A review of Tables 10-17 through 10-19 shows that all aggregated AMOS values are greater than one, indicating that the predicted levels of exposure will not produce adverse health effects in exposed individuals. Although the AMOS values in these tables correspond to the dose received by a 70 kg adult, the AMOS values based on exposures of a 31 kg child are not significantly smaller.

For incinerator emissions, the lowest AMOS values are associated with to exposures to polychlorinated dibenzo-p-

TABLE 10-17

AGGREGATED AMOS VALUES: EXPOSURE TO INCINERATOR EMISSIONS*

Exposure Pathway	Target Organ or System Respiratory	Liver	Reproduction	Kidney	Blood	CNS**
Inhalation	2 E+05	6 E+02	8 E+02	1 E+04	6 E+04	8 E+02
Beef	3 E+07	2 E+03	1 E+02	2 E+02	2 E+02	7 E+04
Milk	5 E+09	2 E+03	4 E+02	2 E+04	3 E+02	3 E+04
Pork	2 E+08	1 E+04	9 E+02	1 E+03	1 E+03	1 E+05
Poultry	2 E+09	1 E+05	9 E+02	4 E+04	5 E+04	1 E+05
Eggs	6 E+06	1 E+03	1 E+02	5 E+06	2 E+06	1 E+03
Produce	3 E+05	3 E+03	7 E+02	3 E+03	3 E+02	1 E+04
Soil Ingestion	2 E+08	1 E+05	3 E+05	3 E+06	3 E+04	1 E+05
Soil Contact	5 E+07	3 E+04	2 E+04	8 E+05	8 E+03	4 E+04
Pond	3 E+06	2 E+04	8 E+03	1 E+04	9 E+02	6 E+04
Cistern	6 E+05	5 E+02	9 E+02	1 E+02	7 E+01	6 E+05

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* Expressed in exponential notation, for example, 4 E+02 = 4 x 10^2, or 400.

** Central Nervous System

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I. AMOS values based on the dose received by a 70 kg adult.

TABLE 10-18

AGGREGATED AMOS VALUES: EXPOSURE TO EVAPORATOR EMISSIONS*

Exposure Pathway	Target Organ or System					
	Respiratory	Liver	Reproduction	Kidney	Blood	CNS**
Inhalation	2 E+05	2 E+04	2 E+05	1 E+05	4 E+03	2 E+05
Beef	3 E+11	1 E+03	1 E+03	3 E+03	5 E+02	3 E+11
Milk	2 E+11	9 E+02	2 E+03	3 E+05	5 E+02	2 E+11
Pork	5 E+10	8 E+03	1 E+04	2 E+04	3 E+03	5 E+10
Poultry	7 E+10	3 E+05	4 E+05	1 E+06	1 E+05	7 E+09
Eggs	1 E+10	7 E+05	9 E+05	2 E+06	3 E+05	1 E+10
Produce	2 E+07	1 E+06	2 E+07	5 E+06	2 E+04	2 E+07
Soil Ingestion	9 E+10	3 E+08	2 E+10	2 E+09	1 E+06	9 E+10
Soil Contact	3 E+10	8 E+07	7 E+09	5 E+08	4 E+05	3 E+10
Pond	2 E+07	2 E+05	2 E+07	3 E+06	3 E+05	2 E+07
Cistern	2 E+10	6 E+06	2 E+08	7 E+06	2 E+06	2 E+10

* Expressed in exponential notation, for example, 4 E+02 = 4 x 10^2, or 400.

** Central Nervous System

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I. AMOS values based on the dose received by a 70 kg adult.

TABLE 10-19

AGGREGATED AMOS VALUES: EXPOSURE TO FUGITIVE EMISSIONS*

Exposure Pathway	Target Organ or System					
	Respiratory	Liver	Reproduction	Kidney	Blood	CNS**
Inhalation	2 E+02	4 E+01	2 E+02	2 E+02	2 E+02	5 E+01
Beef	2 E+9	5 E+03	7 E+08	1 E+08	3 E+08	5 E+03
Milk	4 E+08	2 E+03	2 E+08	3 E+07	7 E+07	2 E+03
Pork	5 E+06	7 E+03	5 E+06	3 E+06	4 E+06	7 E+03
Poultry	6 E+06	1 E+04	6 E+06	4 E+06	6 E+06	1 E+04
Eggs	1 E+07	2 E+04	1 E+07	7 E+06	1 E+07	2 E+04
Produce	3 E+02	1 E+02	3 E+02	3 E+02	3 E+02	2 E+02
Soil Ingestion	3 E+06	1 E+04	3 E+06	2 E+06	3 E+06	1 E+04
Soil Contact	9 E+05	3 E+03	8 E+05	7 E+05	8 E+05	3 E+03
Pond	1 E+04	2 E+03	1 E+04	1 E+04	1 E+04	3 E+03
Cistern	2 E+05	5 E+01	1 E+05	1 E+02	2 E+05	2 E+05

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* Expressed in exponential notation, for example, 4 E+02 = 4 x 10^1, or 400.

** Central Nervous System

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I. AMOS values based on the dose received by a 70 kg adult.

dioxins and dibenzofurans (PCDD/PCDF). AMOS values for these chemicals exceed ten in all cases, indicating that the predicted doses are at least ten times lower than the Acceptable Daily Intake values. For evaporator emissions, the lowest AMOS value is associated with inhalation exposure to 1,1,1-trichloroethane. The lowest AMOS value for fugitive emissions corresponds to inhalation exposure to tetraethyl lead.

It should be noted that the AMOS values reported in this section do not include a consideration of existing levels of contamination of air or soil in the vicinity of the site. As discussed in Chapter 5, a separate assessment was conducted, however, to examine the risks associated with total exposures to the both existing contaminant levels and the contribution associated with the operation of the OWMC Facility. (Data on background contaminant concentrations in soil and air in the vicinity of the site are presented in Chapter 3.) That analysis indicated that AMOS values associated with combined exposure to existing contamination and to facility emissions are greater than one in all cases.

10.4 ACUTE EXPOSURES

10.4.1 METHODOLOGY

Short-term (acute) chemical exposures may occur as the result of on-site and off-site upsets and accidents. For these acute exposures, risk was characterized through a comparison of the predicted dose with an Acute Safe Exposure Limit (ASEL). As discussed in Chapter 9, the ASEL is derived by applying a safety factor to an acute no-observed-effect-level (NOEL). In the risk assessment, the ASEL was compared to the human dose received over a short time period to generate an Adjusted Margin of Safety (AMOS) using the following formula:

$$\mathrm{AMOS} = \frac{\mathrm{ASEL}}{\mathrm{HD}}$$

Where:

AMOS = Adjusted Margin of Safety, unitless

ASEL = Acute Safe Exposure Level, mg/kg

HD = Human Dose, mg/kg

In some cases, it was more appropriate to make a comparison between a predicted concentration and an ASEL based on concentration rather than dose. This procedure was used in situations where the exposure evaluated would be expected to cause a localized irritation effect rather than a systemic effect. For these exposures, the following equation was used:

$$\mathrm{AMOS} = \frac{\mathrm{ASEL}}{C_i}$$

Where:

AMOS = Adjusted Margin of Safety, unitless
ASEL = Acute Safe Exposure Level, mg/m^3 or mg/l
C_i = Chemical concentration, mg/m^3 or mg/l

For acute exposures, an AMOS value less than one indicates that some effect may be observed. However, because of the use of the NOEL rather than the NOAEL in calculating an AMOS, these effects may not be adverse to human health. In such cases, the potential for adverse human health effects was further evaluated using short-term-public-limit (STPL) values based on no-observed-adverse-effect-levels (NOAELs).

In evaluating acute exposures, the doses received by children and by adults were evaluated separately. In most cases, a child will receive a higher dose per kilogram of body weight than will an adult. For example, a 10 year old child weighing 31 kg with a breathing rate of 0.78 m^3/hr during light activity would receive a dose approximately 5% higher than that received by a 70 kg adult performing light work with a breathing rate of 1.7 m^3/hr. It should be noted that the acute exposure limits (ASEL and STPL values) developed for the risk assessment incorporate a safety factor to account for the presence of sensitive individuals, such as children, in the exposed population.

10.4.2 Estimated Risks

The potential public health risks associated with the following upset and accident events were examined in the risk assessment:

On-Site

- A fire in the tank farm storage area for organic waste, resulting in the boiling of halogenated solvents and subsequent volatile emissions;
- Failure of the incinerator stack flue gas scrubbing system, resulting in an increase in the release of acid gases (hydrogen chloride, sulfur dioxide, nitrogen oxides, and hydrogen fluoride);
- Mixing of incompatible wastes in the physical/chemical treatment building, resulting in the release of hydrogen sulfide gas;
- pH excursions during treatment of cyanide wastes, resulting in hydrogen cyanide emissions; and
- pH excursions during sulfide treatment of inorganic wastes, resulting in hydrogen sulfide emissions.

Off-Site

- An off-site spill of waste onto a local access road, resulting in the atmospheric release of volatile organics; and
- An off-site spill of waste into Twenty Mile Creek.

The AMOS values corresponding to the on-site upsets and accidents listed above are presented on Table 10-20. The results of the evaluation of the two off-site spill events are summarized in Tables 10-21 and 10-22.

The results summarized in Table 10-20 indicate that no adverse health effects would not be expected to result from

TABLE 10-20

AMOS VALUES CORRESPONDING TO ON-SITE ACCIDENTS AND UPSETS
(Adults and Children[1])

Event	AMOS (Based on ASEL)	AMOS (Based on STPL)
Tank Farm Fire (adult)	3.7×10^{-2}	0.25
(child)	3.6×10^{-2}	0.25
Incinerator Scrubber Failure		
• Hydrogen Chloride	0.45	1.0
• Hydrogen Fluoride	10.0	--
• Nitrogen Oxides	1.5	--
• Sulfur Dioxide	0.058	1.7
Mixing of Incompatible Waste	2.8	--
pH Excursion During Treatment of Cyanide Bearing Waste	5.9×10^{3}	--
pH Excursion During Sulfide Treatment of Inorganic Waste	100	--

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[1] AMOS values for an adult and a child were calculated for a tank farm fire since ASEL and STPL values for this event were developed in terms of the dose received per kg body weight. For the other events, a single AMOS value for children and adults was calculated, since the ASEL and STPL values were developed in terms of a chemical concentration in air.

AMOS values corresponding to the tank farm fire are based on toxicity data presented in Chapter 9 and dose estimates from Appendix I. AMOS values for the other events are based on toxicity data from Chapter 9 and chemical concentrations in air as calculated in Appendix D.

TABLE 10-21

AMOS VALUES CORRESPONDING TO OFF-SITE SPILL EVENTS
(Adults)

Source of Release	Chemical of Concern	AMOS (Based on ASEL)	AMOS (Based on STPL)
Spill onto the Roadway[1]	Methylene Chloride	5.6×10^{-3}	3.8×10^{-2}
Spill into the Stream[2]	Methylene Chloride	36.2	246
	Pyridine		
	• 230 liter spill		
	- 100 m downstream	5.1×10^{-2}	1.0×10^{-1}
	- 3 000 m downstream	2.8×10^{-1}	5.6×10^{-1}
	- 6 400 m downstream	4.1×10^{-1}	8.2×10^{-1}
	• 1 500 liter spill		
	- 100 m downstream	7.8×10^{-3}	1.6×10^{-2}
	- 3 000 m downstream	4.3×10^{-2}	8.5×10^{-2}
	- 6 400 m downstream	6.3×10^{-2}	1.3×10^{-1}
	• 19 000 liter spill		
	- 100 m downstream	6.2×10^{-4}	1.2×10^{-3}
	- 3 000 m downstream	3.4×10^{-3}	6.8×10^{-3}
	- 5 400 m downstream	5.0×10^{-3}	1.0×10^{-2}

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[1] Dose based on exposure of a 70 kg adult with a breathing rate of 1.7 m^3/h.

[2] Dose based on ingestion of 0.5 liter of water by a 70 kg adult.

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

TABLE 10-22

AMOS VALUES CORRESPONDING TO OFF-SITE SPILL EVENTS
(Children)

Source of Release	Chemical of Concern	AMOS (Based on ASEL)	AMOS (Based on STPL)
Spill onto the Roadway[1]	Methylene Chloride	5.4×10^{-3}	3.7×10^{-2}
Spill into the Stream[2]	Methylene Chloride	32.5	221
	Pyridine		
	• 230 liter spill		
	– 100 m downstream	4.5×10^{-2}	9.1×10^{-2}
	– 3 000 m downstream	2.5×10^{-1}	5.0×10^{-1}
	– 6 400 m downstream	3.6×10^{-1}	7.3×10^{-1}
	• 1 500 liter spill		
	– 100 m downstream	6.9×10^{-3}	1.4×10^{-2}
	– 3 000 m downstream	3.8×10^{-2}	7.5×10^{-2}
	– 6 400 m downstream	5.6×10^{-2}	1.1×10^{-1}
	• 19 000 liter spill		
	– 100 m downstream	5.5×10^{-4}	1.1×10^{-3}
	– 3 000 m downstream	3.0×10^{-3}	6.0×10^{-3}
	– 5 400 m downstream	4.4×10^{-3}	8.8×10^{-3}

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[1] Dose based on exposure of a 31 kg child with a breathing rate of 0.78 m^3/h.

[2] Dose based on ingestion of 0.25 liter of water by a 31 kg child.

Based on toxicity data presented in Chapter 9 and dose estimates from Appendix I.

mixing of incompatible wastes, pH excursions during sulfide treatment, or pH excursions during treatment of cyanide wastes. It was determined, however, that a fire in the tank farm, the failure of the incinerator stack scrubber, and both off-site spill events could pose some level of risk to public health. For the tank farm fire and the off-site spill events, it was found that predicted exposure levels could produce adverse health effects since the short-term-public-limit (STPL) values may be exceeded. The incinerator scrubber failure, however, would be expected to produce only minor reversible effects since the levels of exposure are predicted to be equal to or lower than the STPL values.

In the event of an incinerator scrubber failure, AMOS values based on the ASEL are predicted to be approximately 0.5 for hydrogen chloride and 0.06 for sulfur dioxide. For hydrogen chloride, only slight odors, or minor, reversible irritation effects experienced by sensitive members of the population might be expected (ENVIRON 1985). For sensitive populations exposed to the increased sulfur dioxide emissions, however, it is possible that minor, reversible effects, such as temporary tightness of the chest, might be experienced (ENVIRON 1985).

Exposures to methylene chloride were considered in the evaluations of both the tank farm fire and the transportation spill onto the roadway. In both cases, the AMOS values based on the STPL were estimated to be considerably less than one, indicating that potentially significant health effects would be experienced at the predicted exposure levels. It is difficult to estimate how severe these effects might be since human data on acute exposure to methylene chloride are generally available only for relatively low exposure levels. It can be concluded, however, that potentially severe human health effects could be experienced as a result of these two events, although they are not likely to produce fatalities.

As shown in Tables 10-21 and 10-22, a spill of a "mixer", such as pyridine, into a stream can pose a potentially significant risk to human health. AMOS values based on the STPL are less than one for all exposure scenarios considered in the risk assessment, indicating that potentially significant health effects would be expected. It is difficult to estimate how severe these effects might be, although it is clear that a large spill into Twenty Mile Creek could cause significant stream contamination, and that ingestion of the stream water could result in serious human health effects.

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CHAPTER 11

THE ENVIRONMENTAL RISK ASSESSMENT

11.1 INTRODUCTION

Contaminants released during the operation of the proposed OWMC Facility may pose risks to the health and viability of animals and plant life in the vicinity of the preferred site. It is possible to gain an understanding of the nature and extent of these risks through the application of the principles of risk assessment presented in Chapter 2. Although these principles were developed in the context of human health risk, they can be applied, with proper interpretation, to the assessment of potential adverse impacts on the environment.

The analysis of potential risks to the environment is in some ways far more complex than the analysis of risks to human health. In a human health risk assessment, only one population is considered, although risks to sensitive subgroups within the human population may also be examined. The environment, in contrast, is composed of an enormous array of species inhabiting different niches in one or more ecosystems. Effects on single species--which form the basis for most of the available data on environmental toxicity--do not necessarily reflect the cumulative potential impact on a given ecosystem. Environmental risk assessment is also complicated by a relative dearth of information on the toxicity of chemicals to most environmental species. Data on the toxicity of chemicals to mammalian species, which serve as the basis for human risk assessment, are generally more extensive.

These complexities were considered in developing a methodology for assessing the environmental risks posed by the operation of the proposed OWMC Facility. The objective of the assessment was to evaluate potential adverse effects for key environmental species that may be exposed to emissions from the proposed facility. The analysis was limited to effects on individual species since the current understanding of higher levels of organization, such as ecosystems, is insufficient to support the application of risk assessment.

The general methodology developed to evaluate environmental risks is similar to that described in Chapter 2 for the human health risk assessment, and consists of the following components:

- Identification of selected environmental receptors
- Exposure assessment

- Hazard evaluation
- Risk characterization

These elements of the risk assessment process are discussed in this chapter. Results of the environmental risk assessment are also presented.

11.2 IDENTIFICATION OF ENVIRONMENTAL RECEPTORS

The first step in the environmental risk assessment was an examination of the characteristics of the area surrounding the preferred West Lincoln site. Information specific to the site, including land use, the presence of bodies of surface water, and the presence of specific plant and animal species was compiled and is presented in Chapter 3 and Appendix A of this report. This information was used to identify the range of potential environmental receptors and the critical pathways for exposure to facility emissions.

In analyzing the environmental risks associated with the operation of the proposed OWMC Facility, it is not feasible to evaluate each species that may be present in the site vicinity. Therefore, certain species, referred to in this risk assessment as indicator species, have been selected for analysis. Through the use of conservative assumptions in the assessment, it is believed that adequate protection of these indicator species should provide protection for other environmental receptors as well.

In the risk assessment, the selection of indicator species was based on a consideration of the following factors:

- Prevalence in the site vicinity. In order to reflect the characteristics of the West Lincoln site, consideration was given to those species which are found in the site vicinity.

- Economic and societal value. The selection process was designed to include those organisms which are clearly perceived by the general public as being of economic, recreational, or aesthetic importance.

- Representation of significant aquatic and terrestrial environments. An effort was made to select a range of species that would permit consideration of all potentially contaminated media and all major routes of exposure. Thus, both aquatic and terrestrial species (including those subject to exposure to contaminants via air, soil, and water) were included in the assessment.

- Availability of toxicologic data. While a variety of environmental species may be of concern to the public, toxicologic data specific to many native species are unavailable, and a risk assessment based on such species would provide few meaningful conclusions. The assessment was therefore restricted to those species for which sufficient toxicological data are available.

- Ability to predict potential adverse effects. Indicator species must be characterized sufficiently in the scientific literature to allow for an evaluation of potential pathways for exposure to contaminants released from the OWMC Facility. For example, while adequate data for predicting potential risks may be available for livestock, comparable information for other species, such as reptiles and insects, are not similarly available.

- Relative sensitivity. Once candidate species were selected, toxicological data from the most sensitive species were used in establishing criteria for toxic responses.

The application of these factors in the selection of indicator species involved substantial judgment and balancing to produce a risk assessment that is specific to the proposed facility site, conservative in the determination of potential risks, and capable of incorporating the limited amount of information available for environmental species.

Based on a consideration of the factors outlined above, specific indicator species were selected for evaluation in the environmental risk assessment. These species are listed in Table 11-1.

11.3 EXPOSURE ASSESSMENT

11.3.1 INTRODUCTION

The exposure assessment component of the environmental risk assessment consisted of four steps which parallel those used in the human health risk assessment. These steps were:

- Identification of significant sources of environmental releases;
- Characterization of releases in terms of:
    - frequency and duration;
    - chemicals to be modeled; and
    - magnitude of release;

TABLE 11-1

INDICATOR SPECIES EVALUATED IN THE ENVIRONMENTAL RISK ASSESSMENT

Livestock

- Calf
- Dairy cow
- Steer
- Horse
- Sheep
- Chicken
- Goat
- Pig

Terrestrial Mammals

- Guinea pig
- Hamster
- Mink
- Mouse
- Rabbit
- Rat

Avian Species

- Duck
- Quail
- Heron

Aquatic Species

- Various fish

Crops

- Various plants

- Application of transport models to predict environmental concentrations; and
- Estimation of exposure for the indicator species.

The final step of the exposure assessment, which involved the quantification of exposure to contaminants released from the facility, included considerations specific to the environmental species evaluated in the risk assessment. These considerations reflect critical differences between humans and environmental species relating to diet and other factors which influence the nature and magnitude of potential risks.

11.3.2 CHRONIC EXPOSURES

In the environmental risk assessment, long-term (chronic) exposures were associated with routine incinerator, stack evaporator, and fugitive emissions. Risks associated with exposures to chemicals in leachate generated in the engineered landfill were not considered since contaminant concentrations in ground water were not determined. Long-term exposures were based on maximum average contaminant concentrations in the various environmental media. It was assumed that chronic exposures would be continuous over the lifespan of the plant or animal species.

Those pathways which are believed to pose the greatest risks to potentially exposed environmental receptors have been evaluated in the risk assessment. For example, exposures via inhalation, ingestion of crops, consumption of water, and ingestion of soil were evaluated in assessing risks to cattle. The analysis of potential impacts on fish, on the other hand, was limited to an evaluation of ambient water exposure. The pathways evaluated for each category of environmental species considered in the assessment are summarized in Table 11-2.

For terrestrial animals and birds, the measurement of exposure was the predicted systemic dose, expressed in units of milligram of chemical per kilogram body weight per day (mg/kg/d). The calculation of systemic dose utilizes standard dietary and metabolic assumptions for the indicator species. (These assumptions are presented in Appendix J.) In evaluating exposure of terrestrial animals to dietary contaminants, indicator species were categorized as herbivores, carnivores, or omnivores. Assumptions about the composition of the diet for each of these three groups also appear in Appendix J. Where multiple pathways of exposure were considered, the total dose was obtained by adding the estimated doses from the individual pathways.

For example, in estimating the average daily dose of a particular chemical received by a cow, the concentrations of the chemical in food, water, soil and inspired air were first predicted. Assumptions were then made regarding the weight of

TABLE 11-2

PATHWAYS EVALUATED IN THE ENVIRONMENTAL RISK ASSESSMENT (CHRONIC EXPOSURES)

Livestock	• Direct inhalation • Ingestion of surface water • Ingestion of crops and vegetation • Ingestion of soil
Mammalian Wildlife	• Direct inhalation • Ingestion of surface water • Ingestion of vegetation and/or meat
Crops	• Atmospheric exposure (foliar adsorption and absorption) • Uptake from soil (root adsorption and absorption)
Aquatic Organisms (fish)	• Ambient water exposure
Avian Species	• Direct inhalation • Ingestion of surface water • Ingestion of aquatic organisms

the exposed cow and the daily intake of each of the contaminated media to calculate an average daily dose.

For fish and plants, no average dose was calculated. For these indicator species, exposure was based on the concentration of the chemical in the relevant ambient media. For fish, water was the medium of interest. Plants may be exposed to chemicals present in the soil (typically metals) or in air (typically acid gases or volatile organic chemicals). Since studies addressing both types of exposure are generally unavailable for individual contaminants, the total dose to plants via the two pathways combined was not determined. This is not believed to be a serious limitation since the more significant pathway was evaluated for each chemical examined.

The specific equations and assumptions used to calculate systemic doses for the various indicator species considered in the risk assessment are presented in Appendix J.

11.3.3 ACUTE EXPOSURES

Short-term (acute) exposures are associated with chemical releases caused by accidents or upset conditions. In the environmental risk assessment, short-term releases resulting from an incinerator scrubber failure, a tank farm fire, the mixing of incompatible wastes, and transportation spills were examined. Corresponding acute exposures were based on estimated short-term contaminant emission rates and on the predicted duration of the release.

In assessing on-site accidents and upsets, exposure to chemicals emitted to the atmosphere was examined for livestock and mammalian wildlife (by direct inhalation) and crops (by direct exposure). Since the on-site upset and accident release scenarios considered were of limited duration and involved releases directly to air, exposure pathways other than inhalation were not considered to be significant. Accordingly, those short-term contaminant releases that were considered in the risk assessment are not expected to have a significant adverse impact on fish or avian species.

In the evaluation of off-site transportation accidents, the two types of spills described in Chapter 8 were examined. In the first scenario, the impact of a spill onto a roadway was evaluated for livestock and mammalian wildlife (exposure via direct inhalation) and crops (direct exposure). In the second scenario, direct exposure of aquatic life to chemicals spilled into a stream was examined.

The pathways evaluated in the various upset and accident scenarios examined in the environmental risk assessment are summarized in Table 11-3.

TABLE 11-3

PATHWAYS EVALUATED IN THE ENVIRONMENTAL RISK ASSESSMENT (ACUTE EXPOSURES)

I. On-Site Facility Accidents/Upset Conditions	
Livestock	• Direct inhalation
Mammalian Wildlife	• Direct inhalation
Crops	• Atmospheric exposure (foliar adsorption and absorption)
II. Off-Site Transportation Accident	
Aquatic Organisms (Fish)	• Ambient water exposure
Livestock	• Direct inhalation
Mammalian Wildlife	• Direct inhalation
Crops	• Atmospheric Exposure (foliar adsorption and absorption)

11.3.4 MAJOR ASSUMPTIONS IN THE EXPOSURE ASSESSMENT

A series of conservative assumptions were made in conducting the exposure assessment. For example, environmental exposures for all species were estimated at the point of maximum contamination for each pathway considered. As a result of the use of such conservative assumptions, actual exposures for species present in the vicinity of the site are likely to be significantly less than the exposures estimated in the environmental risk assessment. The major assumptions used in the exposure assessment component of the environmental risk analysis are listed in Table 11-4.

11.4 HAZARD EVALUATION

11.4.1 INTRODUCTION

The first step in the hazard evaluation component of the environmental risk analysis involved the compilation of available toxicity data for the chemicals of concern. These data were reviewed to determine for each indicator species the exposure level at which no adverse effects were observed in the test population. This exposure level is termed the no-observed-adverse-effect level (NOAEL). NOAELs corresponding to short-term (acute) and long-term (chronic) exposures were identified. In cases where all tested exposures produced an

TABLE 11-4

MAJOR ASSUMPTIONS USED IN THE ENVIRONMENTAL RISK ANALYSIS EXPOSURE ASSESSMENT

- Long-term direct exposures were based on the lifespan of the plant or animal.

- For chronic exposures, contaminant concentrations in the various environmental media were based on maximum annual average facility emission rates. It was assumed that exposure begins after 70 years of chemical accumulation in soil and water has occurred.

- Short-term exposures from facility upset conditions and accidents were based on the estimated duration of release.

- Exposures of livestock and wildlife were based on chemical concentrations in air, food crops, vegetation, soil and water, at the point of maximum contamination.

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adverse effect, a lowest-observed- adverse-effect level (LOAEL) provides the best available basis upon which to determine a safe exposure level. (Data complied in the environmental risk assessment are summarized in Appendix L).

The acute or chronic NOAELs (or LOAELs) were divided by appropriate safety factors (which attempt to account for limitations in the available toxicity data), to derive safe systemic doses or safe ambient concentrations. These safe levels of exposure are referred to as criterion doses or concentrations in the environmental risk assessment, and are presented in Appendix L.

With suitable interpretations and modifications, the principles used to evaluate toxicity data in the context of human health risk assessment can be applied to data used to assess risks to exposed environmental species. Certain basic principles and definitions used in this environmental risk assessment, however, require additional clarification. They include distinctions between acute and chronic toxicity, and appropriate measures of toxicity for environmental species.

11.4.2 DEFINING ACUTE AND CHRONIC TOXICITY

One consideration in developing the lowest safe acute and chronic exposure levels (or criterion doses or concentrations) from the available toxicity data is to distinguish between acute and chronic toxicity as they pertain to exposures received in the vicinity of the OWMC Facility. As in assessing human health risk, acutely toxic effects are usually assumed to be associated with exposures of relatively short duration. Chronic effects are associated with exposures of longer duration and are typically more sensitive responses to a chemical exposure. In human health risk assessment, chronic toxicity studies generally involve lifetime or near-lifetime exposure. When evaluating data from ecological toxicity studies, however, distinctions between acute and chronic toxicity are not always clear.

The most important effects in environmental species may be associated with exposures of relatively brief duration, such as developmental effects resulting from exposures occurring during gestation. The responses occurring at the lowest exposure levels in chronic toxicity tests for aquatic organisms are typically effects on sensitive life stages that occur after relatively short, but not acute, exposures and that may be quite severe. For example, aquatic toxicity tests that expose the most sensitive life stage (e.g., larvae) for only 7 to 11 days have now been proposed as equivalent to the longer life-cycle tests (Barnthouse 1986). Given the difficulty in distinguishing between acute and chronic toxicity data for environmental species, it has been recommended that the choice of toxicological data in a risk assessment be based on an analysis of the exposure situation being assessed rather than on arbitrary definitions of acute and chronic toxicity (Barnthouse 1986) .

In the current risk assessment, judgment has been used in defining toxicity data as "acute", "subchronic", or "chronic". For instance, early life stage or life cycle tests are considered to be representative of chronic toxicity for fish, and 24 to 96-hour lethality tests are considered to reflect acute aquatic toxicity. For terrestrial mammals, studies of more than three months are considered to be of sufficient duration for assessing chronic toxicity, while for plants, chronic exposures are assumed to involve those that occur over the majority of a plant's growing season. Studies of intermediate duration are considered subchronic toxicity studies.

11.4.3 RELEVANT MEASURES OF TOXICITY

In assessments of environmental risk, the safe exposure level should ideally be set at a level below which adverse impacts on the health and viability of an environmental species might occur. At issue is the question of which responses constitute appropriate measures of toxicity to various wildlife and agricultural species. Clearly growth, survival, and reproduction for animals and yield for plants are critical measures of environmental toxicity, whereas other measures of toxicity relevant in human risk assessment (e.g., reversible histopathologic changes or biochemical alterations) are of less certain environmental significance. Because of limitations in the available literature, however, it is difficult, if not impossible, to base an environmental risk assessment on only those measures of toxicity that are clearly critical. Therefore, assessment of inherent hazard has been conservatively based on adverse effects occurring at the lowest exposure levels, whether or not these effects are associated with life-shortening or reproductive impairment. Support for this practice is found in several analyses of wildlife toxicity data (Tucker and Leitze 1979; Mayer et al. 1986).

Only threshold effects were considered in evaluating the inherent hazard of the chemicals of concern in the OWMC environmental risk assessment. By definition, threshold effects are those which are not expected to occur unless some minimum (threshold) level of exposure is reached. Carcinogenicity, which is generally considered a no-threshold effect, was not considered in evaluating the inherent hazard of the chemicals of concern because no generally accepted methods for assessing cancer risk levels in animals have been developed. Furthermore, while tumors are known to occur in fish and certain livestock species, no information on the carcinogenic potential of the chemicals of concern to these species was found in the literature. The only available data on carcinogenic potency of the chemicals of concern are data in experimental laboratory animals. Approaches for evaluating no-threshold effects in an environmental risk assessment are currently being investigated.

11.4.4 DERIVATION OF THE CRITERION DOSE OR CONCENTRATION

The criterion dose or concentration was defined in the environmental risk assessment as the lowest safe systemic dose or safe ambient concentration. This dose or concentration was calculated for each contaminant for each of the indicator species for which toxicity data were available. Safe systemic doses were derived from experimental NOAELs or LOAELs by applying uncertainty or safety factors:

$$CD = \frac{NOAEL}{SF}$$

Where:

CD = Criterion Dose, mg/(kg-d)
NOAEL = No Observed Adverse Effect Level, mg/(kg-d)
SF = Safety Factor

In some cases, it was necessary to use a lowest-observed-adverse-effect-level (LOAEL) rather than a NOAEL to calculate the criterion dose due to limitations in available data. Criterion concentrations for plants and fish were determined using a similar approach. Separate criterion doses or concentrations were determined to evaluate acute and chronic exposures using appropriate toxicity data and safety factors.

As in the human risk assessment, the application of safety or uncertainty factors to the available data addresses limitations in the available toxicity data. Safety factors were used to account for such limitations as interspecies differences in sensitivity and experimental exposures of duration less than those predicted. In the field of human risk assessment, guidelines for the use of uncertainty factors to derive acceptable daily intakes (ADIs) have been adopted by several governmental bodies and scientific organizations such as the U.S. Environmental Protection Agency and National Research Council of the National Academy of Sciences (Dourson and Stara 1983). Because the science of environmental risk assessment is in its infancy, similar guidelines for the use of uncertainty factors in deriving safe exposure levels have not been rigorously researched or widely accepted by the scientific community. The uncertainty factors developed in this environmental risk assessment, as well as empirical support for selection of these safety factors, are presented in Appendix K.

In the derivation of criterion doses for terrestrial mammals and birds from the available experimental data, units of exposure have been converted (as necessary) to a corresponding systemic dose in units of milligram of chemical per kilogram body weight per day (mg/kg/d). Conversion of exposure levels to systemic dose (in mg/kg/d) is necessary because experimental testing involves exposures of varying durations (e.g., 6 hours per day versus continuous exposure) and via a number of routes (e.g., inhalation, ingestion, and drinking water) for which units of exposure are not directly comparable. For fish and plants, the toxicity of a chemical is typically assessed in terms of the ambient concentration of a toxicant; thus no conversion of exposure units is required to derive criterion concentrations for these indicator species.

It should be recognized that the data base with respect to environmental toxicity is sparse for many of the chemicals of concern. To provide some indication of potential risk posed by these chemicals to plants and animals for which no toxicological data are available, criterion doses (in units of mg/kg/d or, as appropriate, mg/m^3 or mg/l) were assumed to be the same in related species. The application of criterion doses to related indicator species was limited to the following situations:

- If toxicity data for livestock were not available, the lowest criterion dose from an experimental animal (i.e., guinea pig, mink, mouse, rabbit, rat) was extrapolated to the dairy cow (as representative of livestock species).
- If toxicity data for the heron were not available, the lowest criterion dose from other bird species (i.e., chicken, duck, quail) was extrapolated to the heron.

It should be noted that additional uncertainty is introduced by such extrapolation of toxicity data in the environmental risk assessment.

11.5 ENVIRONMENTAL RISKS ASSOCIATED WITH THE OPERATION OF THE OWMC FACILITY

11.5.1 INTRODUCTION

The final step in the environmental risk assessment was to combine the information developed during the hazard evaluation with the dose estimates to provide an overall assessment of potential risk to plants and animals. This step, known as risk characterization, was accomplished by determining the adjusted margin of safety (AMOS) for individual chemicals and indicator species. As defined in the environmental risk assessment, the AMOS is the ratio of the acceptable criterion dose or concentration for a species to the predicted systemic dose or ambient concentration:

$$\text{AMOS} = \frac{\text{CD}}{\text{PD}}$$

Where:

AMOS = adjusted margin of safety, unitless

CD = criterion dose (mg/kg or concentration (mg/l or mg/m^3)

PD = predicted dose (mg/kg) or concentration (mg/l or mg/m^3)

The procedure used for deriving AMOS values in the environmental risk assessment was consistent with that used for determining AMOS values associated with chronic exposures to non-carcinogens in the human health risk assessment.

The smaller the predicted AMOS value, the greater the potential for environmental risks. In the environmental risk assessment, an AMOS of less than 1 was considered to represent a level of potential environmental concern. While some level of risk may be associated with any exposure to a chemical, the risks corresponding to an AMOS value of 1 or greater are not considered to be of significant environmental concern.

Since the derivation of an AMOS value cannot fully address the many uncertainties inherent in risk assessment, evaluating the hazards of chemicals in the environment should be considered an iterative process. Thus, when a conservative analysis suggests that a surrogate chemical presents some level of potential environmental concern (i.e., AMOS < 1), the assumptions used in the analysis should be re-evaluated before making a judgment on the degree of risk posed by the chemical to agricultural or wildlife species.

In this section, the environmental risks associated with the operation of the OWMC Facility are presented.

11.5.2 RISKS ASSOCIATED WITH CHRONIC EXPOSURES

11.5.2.1 Introduction

As previously discussed in Section 11.3.2, the following sources of routine emissions were examined in the environmental risk assessment:

- Incinerator stack emissions;
- Evaporator stack emissions; and
- Fugitive releases.

Exposures were estimated both with and without a consideration of existing levels of chemicals in the Welland River.

11.5.2.2 Incinerator Stack Emissions

The results of the environmental risk assessment indicate that routine incinerator emissions should not pose a risk to exposed plant and animal species. AMOS values for all combinations of chemicals and species were greater than one; for over 75% of the chemical/species combinations examined, AMOS values exceeded 1000. In a very few cases, AMOS values corresponding to the exposure of an indicator species to a specific chemical were less than 10 (e.g., for lead and the dairy cow, and for SO_2 and the guinea pig). Even for these chemicals, however, incinerator emissions would not be expected to produce adverse effects in plants or animals.

11.5.2.3 Evaporator Stack Emissions

As with incinerator stack emissions, routine releases from the evaporator stack (as well as those associated with make-up water to the incinerator scrubbing system) should not pose a risk to exposed plant and animal species. In all cases, AMOS values were greater than one; for most combinations of indicator species and contaminants the AMOS exceeded one by many orders of magnitude. None of the calculated AMOS values were less than 10, suggesting that the potential for adverse effects is extremely small.

11.5.2.4 Fugitive Emissions

As with releases from incinerator and evaporator, routine fugitive emissions from the OWMC Facility would not be expected to pose a risk to exposed plant and animal species. AMOS values for all combinations of chemical contaminants and indicator species considered exceeded one; for approximately 75 percent of the chemical/indicator species combinations, AMOS values were 1000 or higher. For a few chemical/indicator species combinations examined, the AMOS approached one (e.g., the AMOS for toluene and the mouse, and for formaldehyde and plants).

11.5.2.5 Existing Contamination of the Welland River

As discussed in Chapter 3, available monitoring data show that the Welland River currently contains relatively high levels of certain metals and phenol. An analysis has therefore been performed to determine whether emissions from the proposed OWMC Facility could substantially contribute to existing background levels of these contaminants in the Welland River and thereby lead to adverse effects in exposed environmental species. In this analysis, existing chemical concentrations in the Welland River were added to the levels associated with the operation of the proposed facility.

The contribution of metals and phenol from routine OWMC emissions to the Welland River are very small compared to existing background concentrations. For example, concentrations of metals in the Welland River resulting from evaporator emissions were predicted to be at least 16 orders of magnitude (1×10^{16}) smaller than measured background concentrations; metals concentrations in the river resulting from incinerator emissions are expected to be at least 11 orders of magnitude (1×10^{11}) smaller than background. Fugitive releases from the facility are not expected to introduce any appreciable quantities of metals into the environment. Emissions from the proposed facility are therefore not expected to significantly contribute to existing background levels of metals or phenol in the Welland River, and thus are not expected to produce any adverse impacts on aquatic life or on animals that use the river as a source of food or drinking water.

The results of this evaluation suggest, however, that existing metal concentrations in the Welland River may pose a risk to fish and water fowl (modeled here as the heron). Those metal/indicator species combinations for which AMOS values are less than one are listed in Table 11-5. Table 11-5 also presents the ratio of the systemic dose to the criterion dose (1/AMOS), which provides another measure of the difference between the estimated dose (or ambient concentration) and the criterion dose (or concentration). As this table shows, criterion doses or concentrations were exceeded for fish or the heron for eight metals.

The existing concentrations of certain metals in the Welland River can also be compared to water quality criteria established by the Ontario Ministry of the Environment (OMOE 1978) and the United States Environmental Protection Agency (USEPA 1986). The criteria for the metals detected in the Welland River are shown in Table 11-6. As shown, the OMOE criteria for cadmium and mercury are exceeded by existing levels in the Welland River; USEPA criteria for lead and mercury are exceeded. (The OMOE criterion for lead would also be exceeded by existing levels if alkalinity in the Welland

TABLE 11-5

MARGINS OF SAFETY CORRESPONDING TO BACKGROUND LEVELS OF CERTAIN METALS IN THE WELLAND RIVER[1]

Contaminant	Indicator Species	AMOS	Ratio of Systemic Dose to Criterion Dose (1/AMOS)
Arsenic	Heron	6.5×10^{-1}	1.5
Cadmium	Fish	6.7×10^{-2}	14.9
	Heron	9.5×10^{-2}	10.5
Chromium	Fish	2.4×10^{-1}	4.2
Copper	Fish	2.5×10^{-2}	40.0
	Heron	1.2×10^{-1}	8.3
Lead	Fish	2.8×10^{-2}	35.7
Mercury	Fish	4.3×10^{-1}	2.3
	Heron	1.3×10^{-2}	76.9
Nickel	Fish	5.2×10^{-1}	1.9
Zinc	Fish	2.0×10^{-1}	5.0
	Heron	5.0×10^{-2}	20.0

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[1] Concentrations of metals in the various facility emissions are predicted to be zero or many orders of magnitude lower than background concentrations measured in the Welland River. Thus, results of the analyses for incinerator stack emissions, evaporator stack emissions, and fugitive releases for the metals are essentially the same.

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose.

TABLE 11-6

EVALUATION OF EXISTING METAL CONCENTRATIONS IN THE WELLAND RIVER USING OMOE AND USEPA WATER QUALITY CRITERIA

Chemical	OMOE (1978) Criteria (mg/L)	USEPA (1986) Criteria (mg/L)[1]	AMOS based on EPA Criteria[1]	AMOS based on OMOE Criteria[1]
Arsenic	1.0×10^{-1}	4.8×10^{-2}(2)	16	33
Cadmium	2.0×10^{-4}	1.1×10^{-3}(3)	1.1	0.2
Chromium (VI)	1.0×10^{-1}	1.1×10^{-2}	1.1	10
Copper	5.0×10^{-3}	1.2×10^{-2}(3)	1.2	0.5
Lead	2.5×10^{-2}(4)	3.2×10^{-3}(3)	0.4	2.8
Mercury	2.0×10^{-4}	1.2×10^{-5}	0.3	0.5
Nickel	2.5×10^{-2}	9.6×10^{-2}(3)	14	3.6
Zinc	3.0×10^{-2}	4.7×10^{-2}	2.8	1.8

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1 Based on concentrations resulting from incinerator stack emissions plus background concentrations in the Welland River.

2 USEPA found data insufficient to develop criteria. Value presented is the lowest-observed-effect level for a freshwater fish.

3 USEPA criterion is dependent on water hardness. Value presented assumes a hardness of 100 mg/L.

4 OMOE criterion is dependent on alkalinity. Value presented assumes an alkalinity of greater than 80 mg/L as $CaCO_3$. The lowest lead criterion, 5.0×10^{-3} mg/L, corresponds to an alkalinity of less than 20 mg/L.

River is less than 20 mg/L as $CaCO_3$) Background concentrations of arsenic, chromium, copper, nickel, and zinc are lower than both OMOE and USEPA water quality criteria, although they exceed the criterion concentrations derived for the risk assessment.

In the evaluation of potential effects of background concentrations of metals in the Welland River, total metals concentrations in the water column (i.e., dissolved plus suspended) were used. This conservative practice may have overestimated the amount of metals available to aquatic species, and thus may have led to overestimates of the level of risk. It is possible that the levels of dissolved metals in the Welland River, which may be a more appropriate basis for assessing environmental risk, occur at levels that do not present a risk of adverse effects to fish life.

11.5.3 RISKS ASSOCIATED WITH ACUTE EXPOSURES

11.5.3.1 Introduction

The following upset and accident scenarios were evaluated for their potential to lead to adverse effects in exposed livestock, crops, and wildlife:

On-Site

- The failure of the scrubbing system designed to control acid gas emissions from the incinerator stack.
- A fire in the organic waste tank farm resulting in the boiling of a storage tank containing solvent wastes.
- The mixing of incompatible wastes.
- pH excursions during the treatment of cyanide wastes, resulting in the release of hydrogen cyanide gas.
- pH excursions during the sulfide treatment of inorganic wastes, resulting in the release of hydrogen sulfide gas.

Off-Site

- A 18 tonne tank truck transporting a load of liquid wastes is involved in a collision resulting in a tank rupture and the release of material onto the roadway.
- An equipment malfunction or transportation accident results in the release of liquid waste directly into Twenty Mile Creek.

The upset and accident scenarios are described in Chapters 7 and 8, and in Appendix C.

In the risk assessment, the selection of the chemicals for analysis in the upset and accident scenarios has been based, in part, on acute mammalian toxicity (ENVIRON 1985). While this may be an appropriate basis for livestock and terrestrial mammals, it is possible that the selection process may have failed to include chemicals of high acute toxicity to plants, fish, or birds. This limitation should be recognized in interpreting the results of the assessment.

The results of the environmental risk assessment for the on-site upset and accident scenarios examined are presented below in the following subsections.

11.5.3.2 Incinerator Scrubber Failure

Results of the assessment of environmental risks associated with the incinerator scrubber failure are presented in Table 11-7. AMOS values for SO_2, NO_x, HCl, and HF are greater than one for all environmental species examined, indicating that exposures to these acid gases are not expected to have any adverse environmental impacts. The lowest AMOS values are for HCl and HF and plants, indicating that vegetation may be more sensitive to these gases than animals.

11.5.3.3 Tank Farm Fire

Results of the assessment of environmental risks associated with the tank farm fire are presented in Table 11-8. As shown in the table, AMOS values for the dairy cow and mouse are less than one; AMOS values for the guinea pig and rat are slightly greater than one. These results suggest that this upset event could pose some level of risk to some livestock and wildlife species. A review of the toxicity data for methylene chloride suggests that the predicted exposure levels would not cause

TABLE 11-7

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH AN INCINERATOR SCRUBBER FAILURE

	Acute Environmental Risks			
	Selected Species	Criterion Dose (mg/kg/day)	Predicted Dose (mg/kg/day)	AMOS
Sulfur Dioxide	Mouse	1.1	2.6×10^{-3}	4.2×10^{2}
	Dairy cow	1.1(1)	4.3×10^{-4}	2.6×10^{3}
Nitrogen Oxides	Guinea pig	2.9×10^{-2}	2.7×10^{-5}	1.1×10^{3}
	Mouse	2.2×10^{-3}	7.6×10^{-5}	29.0
	Dairy cow	2.2×10^{-3}(1)	1.3×10^{-5}	1.7×10^{2}
Hydrogen Chloride	Mouse	1.2×10^{-2}	3.4×10^{-3}	3.5
	Dairy cow	1.2×10^{-2}(1)	5.6×10^{-4}	21.0
	Plant	6.1×10^{-2} mg/m^3	2.0×10^{-3} mg/m^3	30.5
	Tomato	2.0×10^{-2} mg/m^3	2.0×10^{-3} mg/m^3	10.0
	Buckwheat	2.9×10^{-3} mg/m^3	2.0×10^{-3} mg/m^3	1.5
Hydrogen Fluoride	Calf	9.0×10^{-1}	6.8×10^{-6}	1.3×10^{5}
	Goat	2.0	5.2×10^{-6}	3.8×10^{5}
	Guinea pig	9.0×10^{-1}	2.7×10^{-5}	3.3×10^{4}
	Rabbit	1.1	3.4×10^{-5}	3.2×10^{4}
	Steer	5.8×10^{-1}	1.1×10^{-5}	5.3×10^{4}
	Plant	9.0×10^{-5} mg/m^3	4.5×10^{-5} mg/m^3	2.0

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[1] Acute toxicity data for livestock species were not available. Therefore, a criterion dose for the dairy cow was derived from the criterion dose for the most sensitive laboratory animal.

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose. Exposure period of 30 seconds assumed (see Appendix C).

TABLE 11-8

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH A TANK FARM FIRE

	Acute Environmental Risks			
	Selected Species	Criterion Dose (mg/kg/day)	Predicted Dose (mg/kg/day)	AMOS
Methylene Chloride	Dairy cow	$3.0 \times 10^{1(1)}$	3.2×10^{1}	0.94
	Guinea pig	1.2×10^{2}	6.9×10^{1}	1.74
	Mouse	3.0×10^{1}	1.9×10^{2}	0.16
	Rat	1.4×10^{2}	6.9×10^{1}	2.02
	Alfalfa	3.0×10^{4} mg/m^3	1.1×10^{2} mg/m^3	2.6×10^{2}

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[1] Acute toxicity data for livestock species were not available. Therefore, a criterion dose for the dairy cow was derived from the criterion dose for the most sensitive laboratory animal (i.e., the mouse).

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose. Exposure period of 2.6 hours assumed (see Appendix C).

irreversible damage to livestock or wildlife, although short-term effects might be observed.

The most sensitive effects associated with methylene chloride exposure, and those most likely to be observed as a result of exposures from a tank farm fire, are those on the nervous system and on behavior. Exposure to 3 474 mg/m^3 of methylene chloride (approximately three times the maximum off-site concentration expected in the event of a tank farm fire) for three hours decreased the spontaneous activity of mice (USEPA 1985). Effects other than those on the nervous system have been observed only at acute methylene chloride concentrations higher than those predicted in the tank farm fire scenario. Inhalation exposure of mice to 17 000 mg/m^3 caused liver damage (histological changes of the liver), but only after 12 hours of exposure (USEPA 1985). Depression of cardiac function has only been reported at concentrations in excess of 69 000 mg/m^3 (USEPA 1985). Brain death has occurred in rats exposed to 93 800 mg/m^3 for 1.5 hours (USEPA 1985), an exposure level substantially higher than that predicted in the current scenario (1 060 mg/m^3). Thus, based on the available acute toxicity data for methylene chloride in experimental animals, one would expect symptoms associated with temporary depression of the central nervous system, but no irreversible effects in livestock and terrestrial wildlife.

11.5.3.4 Mixing of Incompatible Wastes

Table 11-9 summarizes the results of the assessment of short-term environmental risks associated with an upset scenario involving the mixing of incompatible wastes to produce hydrogen sulfide gas. AMOS values for the species evaluated in this scenario range from 25 to 4 000. These AMOS values strongly suggest that the mixing of incompatible wastes is unlikely to pose any environmental risks.

11.5.3.5 pH Excursions, Treatment of Cyanide Wastes

Table 11-10 summarizes the evaluation of potential acute environmental risks associated with a pH excursion during treatment of cyanide bearing wastes. It should be noted that only limited data with respect to the acute toxicity of hydrogen cyanide to plants and animals were available. The data that were examined, however, indicate that the potential for adverse effects on environmental receptors is very low.

11.5.3.6 pH Excursions, Sulfide Treatment

The evaluation of potential risks to plants and animals resulting from a pH excursion during sulfide treatment is summarized in Table 11-11. As shown in the table, all AMOS values exceed 500, an indication that the event is highly unlikely to pose a significant risk to the environment.

TABLE 11-9

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH THE MIXING OF INCOMPATIBLE WASTES

	Acute Environmental Risks			
	Selected Species	Criterion Dose (mg/kg/day)	Predicted Dose (mg/kg/day)	AMOS
Hydrogen Sulfide	Chicken	1.6	6.9×10^{-3}	2.3×10^{2}
	Dairy cow	4.7×10^{-2}(1)	1.9×10^{-3}	2.5×10^{1}
	Mouse	6.3×10^{-1}	1.2×10^{-2}	5.3×10^{1}
	Pig	4.7×10^{-2}	5.6×10^{-4}	8.4×10^{1}
	Rabbit	2.1×10^{1}	5.3×10^{-3}	4.0×10^{3}
	Rat	1.8×10^{-1}	4.2×10^{-3}	4.3×10^{1}
	Tomato	2.1×10^{-1} mg/m^3	6.9×10^{-3} mg/m^3	3.0×10^{1}

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[1] A criterion dose for the dairy cow was derived from the criterion dose for the most sensitive animal species tested (i.e., the pig).

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose. Exposure period of approximately 40 minutes assumed.

Table 11-10

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH A pH EXCURSION DURING THE TREATMENT OF CYANIDE BEARING WASTE

	Acute Environmental Risks			
	Selected Species	Criterion Dose (mg/kg/day)	Predicted Dose (mg/kg/day)	AMOS
Hydrogen Cyanide	Dairy cow	6.7×10^{-2}(1)	2.0×10^{-6}	3.3×10^{4}
	Mouse	6.7×10^{-2}	1.2×10^{-5}	5.6×10^{3}

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[1] Acute toxicity data for livestock species were not available. Therefore, a criterion dose for the dairy cow was derived from the criterion dose for the most sensitive laboratory animal (i.e., the mouse).

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose. Exposure period of 1 hour assumed.

Table 11-11

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH A pH EXCURSION DURING SULFIDE TREATMENT OF METALS

	Acute Environmental Risks			
	Selected Species	Criterion Dose (mg/kg/day)	Predicted Dose (mg/kg/day)	AMOS
Hydrogen Sulfide	Chicken	1.6	3.0×10^{-4}	5.3×10^{3}
	Dairy cow	4.7×10^{-2}(1)	8.3×10^{-5}	5.7×10^{2}
	Mouse	6.3×10^{-1}	5.0×10^{-4}	1.3×10^{3}
	Pig	4.7×10^{-2}	2.4×10^{-5}	2.0×10^{3}
	Rabbit	2.1×10^{1}	2.3×10^{-4}	9.1×10^{4}
	Rat	1.8×10^{-1}	1.8×10^{-4}	1.0×10^{3}
	Tomato	2.1×10^{-1} mg/m^{3}	3.0×10^{-4} mg/m^{3}	7.0×10^{2}

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1 Acute toxicity data for the dairy cow was not available; therefore, a criterion dose for the dairy cow was derived from the criterion dose for the most sensitive animal species tested (i.e., the pig).

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose. Exposure period of 1 hour assumed.

11.5.3.7 Spill onto the Roadway

Results of the environmental risk assessment of a transportation spill of methylene chloride onto a roadway are presented in Table 11-12. As shown in this table, the AMOS values for the dairy cow, guinea pig, mouse, and rat are less than one; AMOS values range from 0.023 to 0.30. These results suggest that this scenario poses some level of risk to environmental receptors under the conditions of exposure modeled in this assessment (i.e., exposure for one hour at a distance of 20 meters from the site of the accident). As discussed in Section 11.5.3.3, these effects might include symptoms associated with temporary depression of the central nervous system, but no irreversible effects in livestock or terrestrial wildlife. An AMOS value of 39 for alfalfa would suggest that crops and other plant life are not as sensitive to short-term exposures to methylene chloride as are mammalian species.

11.5.3.8 Spill into a Stream

As previously discussed in Chapter 8, the behavior of three general types of chemicals following a spill into Twenty Mile Creek was evaluated: (1) "mixers", soluble substances that mix rapidly with water; (2) "sinkers," relatively immiscible substances that sink to the bottom of a watercourse; and (3) "floaters", immiscible substances that float on the water surface. In the environmental risk assessment, the possible

TABLE 11-12

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH TRANSPORTATION SPILL OF METHYLENE CHLORIDE ONTO THE ROADWAY[1]

Selected Species	Criterion Dose (mg/kg/day)	Predicted Dose (mg/kg/day)	AMOS
Dairy cow	3.0×10^{1}(2)	2.1×10^{2}	0.14
Guinea pig	1.2×10^{2}	4.6×10^{2}	0.26
Mouse	3.0×10^{1}	1.3×10^{3}	2.3×10^{-2}
Rat	1.4×10^{2}	4.6×10^{2}	0.30
Alfalfa	3.0×10^{4} mg/m^3	7.7×10^{2} mg/m^3	39

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[1] The scenario assumes that ambient air concentrations of methylene chloride resulting from a transportation spill are 18.4 g/m^3 and that exposures last for 1 hour.

[2] Acute toxicity data for livestock species were not available. Therefore, a criterion dose for the dairy cow was derived from the criterion dose for the most sensitive laboratory animal (i.e., the mouse).

See Appendix J for the derivation of the predicted dose and Appendix L for the basis of the criterion dose. Exposure period of 1 hour assumed.

impacts of a spill of these types of special wastes on freshwater aquatic life has been quantitatively evaluated. Potential adverse effects on terrestrial wildlife or livestock have not been quantitatively assessed, although qualitative conclusions can be drawn.

The "mixer" selected for analysis in this assessment was pyridine (see Appendix B for a description of the procedure used to select chemicals for evaluating the risks associated with a spill into a stream). The acute aquatic criterion concentration for pyridine was calculated to be 0.5 mg/l*; exposures to levels greater than this concentration would be likely to have some adverse effects on freshwater aquatic life.

As shown in Table 11-13, contaminant levels resulting from both average (approximately 230 liter) and large (approximately 19 000 liter) spills consisting entirely of pyridine greatly exceed the acute aquatic criterion concentration, even at a distance 6 400 meters downstream of the spill. In fact, predicted concentrations for both average and large spills exceed the reported 96-hour LC_{50}** for the carp (26 mg/l).

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* Based on a 96-hour LC_{50} value in the carp of 26 mg/l (Rao _et al_. 1975) and the application of a safety factor of 50 (see Appendix K).

** The LC_{50} is the concentration expected to be lethal to 50 percent of the exposed population.

TABLE 11-13

SHORT-TERM ENVIRONMENTAL RISKS ASSOCIATED WITH A SPILL OF SPECIAL WASTE INTO TWENTY MILE CREEK

Contaminant	Spill Size	
	230 liter	19000 liter
• "Mixer" (Pyridine)		
-- Concentration 100 m downstream (mg/l)	541	45 112
-- Concentration 6 400 m downstream (mg/l)	68	5 639
-- Acute aquatic criterion concentration (mg/l)	0.5	0.5
• "Sinker" (Methylene Chloride)		
-- Concentration just downstream of the concentration zone (mg/l)	10	NA(1)
-- Acute aquatic criterion concentration (mg/l)	4	NA(1)
• "Floater" (Petroleum Distillates)	NA(2)	NA(2)

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(1) Not applicable; large spills of "sinkers" may behave more like spills of "mixers" due to the small volumetric flow of Twenty Mile Creek.

(2) Not applicable; "floaters" are not expected to mix with the water column, and thus would not directly effect aquatic life in the stream.

The approach used to calculate chemical concentrations in stream water is described in Appendix E. Acute aquatic criterion concentrations are taken from Appendix L.

While aquatic life would not be exposed to the modeled concentrations presented in Table 11-13 for as long as 96 hours (i.e., the duration of exposure in the acute toxicity study), it is still likely that fish kills could occur as a result of a spill such as that modeled in the current scenario. The same investigators (Rao et al. 1975) reported a 24-hour LC_{50} of 47.5 mg/l, suggesting that exposures of shorter duration do not markedly increase the LC_{50} for pyridine.

Exposures of terrestrial wildlife and livestock to stream water contaminated by a spill of pyridine have not been quantitatively evaluated in the risk assessment. Based on the predicted pyridine concentraions, however, it can be assumed that ingestion of the contaminated water would likely have an adverse impact on both wildlife and livestock.

The "sinker" selected for evaluation was methylene chloride; the acute aquatic criterion concentration for this chemical was calculated to be 4 mg/l.* An average (approximately 230 L) spill of methylene chloride into Twenty Mile Creek would result in a stream concentration just downstream from the zone of contamination of approximately 10 mg/l. This stream concentration exceeds by 2.5-fold the acute criterion concentration for methylene chloride, suggesting that the spill modeled in this assessment could have some acute effects on aquatic life near the spill. Since the lowest reported LC_{50} value for methylene chloride in fish is 19 times higher than the predicted concentration in Twenty Mile Creek, it is not expected that a spill of methylene chloride as modeled in the current scenario would result in a fish kill.

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* Based on an LC_{50} value of 190 mg/l in the fathead minnow (USEPA 1985) and the application of a safety factor of 50 (see Appendix K).

As discussed in Appendix E, larger spills would tend to behave more as a "mixer," and were therefore not evaluated. It can be assumed, however, that larger spills could have a more significant impact on aquatic life.

The "floater" modeled in this assessment is petroleum distillates. Unlike "sinkers" and "mixers", which become mixed in the water column, "floaters" form a separate, visible, contaminant layer on the water surface. Aquatic toxicity data is generally expressed in terms of a concentration in the water, and because petroleum distillates are not expected to mix with the water column, no direct toxicity to fish or other aquatic life is expected. A spill of petroleum distillates might indirectly affect aquatic life, however, by reducing oxygen levels in the stream. It has been estimated that in the event of a 19 000 liter spill, 100% of the "floater" selected for evaluation, petroleum distillates, would volatilize before reaching Jordan Harbor, a distance of approximately 6 000 meters.

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CHAPTER 12

CONCLUSIONS AND RECOMMENDATIONS

12.1 INTRODUCTION

The general methodology used in the risk assessment for the proposed OWMC Hazardous Waste Management Facility has been presented in the previous chapters of this report. Four principal categories of contaminant release were examined: (1) routine atmospheric emissions associated with normal facility operations; (2) the migration of leachate from the engineered landfill; (3) short-term releases resulting from on-site upsets and accidents; and (4) short-term releases resulting from off-site transportation accidents and malfunctions.

In this chapter, the results of the human health and environmental risk assessments are discussed, and recommendations for risk management strategies based on these results are provided.

12.2. ROUTINE ATMOSPHERIC EMISSIONS

12.2.1 HUMAN HEALTH RISK ASSESSMENT

The human health risks associated with exposure to routine atmospheric emissions were evaluated in Section 10.2. The following conclusions can be drawn from the results of that assessment:

- The release of non-carcinogens to the atmosphere during normal operation of the proposed facility is not likely to produce adverse health effects in exposed individuals.
- Cancer risks associated with exposures to incinerator, evaporator, and fugitive emissions are not likely to exceed one in one million, i.e., 1 x 10^{-6}, for any pathway.
- The most significant exposure pathways for incinerator emissions were predicted to be direct inhalation and the ingestion of meat and milk. For evaporator emissions, ingestions of meat, milk, and crops were predicted to be the most significant pathways for human exposure. For fugitive releases, direct inhalation and ingestion of crops were determined to be most potentially significant.
- The individual cancer risks associated with exposures to incinerator and fugitive emissions were predicted to be of approximately the same magnitude. Risks associated with exposures to evaporator emissions were estimated to be significantly lower than those associated exposures to with incinerator and fugitive emissions.

As discussed in Chapter 10, a number of conservative assumptions likely to result in an overestimate of risk were made in the assessment. For example, exposures to emissions were assumed to occur for a period of 70 years at the location of maximum contamination.

It should be noted, however, that there are significant uncertainties associated with the prediction of chemical exposures, especially through the consumption of food. Data and methodologies for estimating the accumulation of chemicals in plants, meat, and milk are limited. In most cases, such accumulation must be predicted using generalized correlations rather than experimental data for individual chemicals (see Appendix D).

It should also be recognized that the actual level of risk during facility operations will be dependent, in part, on the nature and magnitude of the routine atmospheric emissions. In the risk assessment, conservative approaches likely to overestimate risk have been adopted in predicting routine emissions. Data used in developing these emissions estimates, however, are limited in some cases, particularly for the evaporator.

12.2.2 ENVIRONMENTAL RISK ASSESSMENT

The risks to plants and animals exposed to routine atmospheric emissions are presented in Section 11.5.2. As discussed in that section, the predicted levels of exposure are not likely to produce adverse effects to crops, livestock, or wildlife in the vicinity of the proposed site. Existing levels of certain contaminants in the Welland River, however, may already pose a significant risk to fish and animals for which fish are a primary food source.

The evaluation of environmental risk was necessarily limited by a number of factors. These limitations included: (1) an incomplete knowledge of the toxicity of the emissions to certain environmental receptors; (2) limited information with which to develop accurate estimates of exposure; (3) a limited understanding of the potential effects of contaminants on ecosystems (as opposed to single species); and (4) a limited knowledge of the possible interactions of mixtures of chemicals in the environment and their net effects on environmental receptors. To account for these limitations, conservative assumptions were made with respect to toxicity and exposure.

12.2.3 RISK COMPARISONS

The routine atmospheric release of non-carcinogens from the proposed OWMC Facility is not predicted to pose a significant risk to public health or the environment. The exposure levels expected during normal operation of the facility will be significantly lower than those that would be expected to produce adverse effects in humans, plants, or animals.

As discussed in Chapter 9, however, exposures to carcinogens are not typically evaluated through a simple comparison to a safe "threshold" dose. It is often assumed for carcinogens that some risk is associated with even the smallest levels of exposure. Thus, it is necessary for individuals, communities, and regulatory bodies to make judgments as to what level of incremental cancer risk might be considered "insignificant" or "acceptable." In making such judgments, it is often helpful to make comparisons to risks encountered in everyday life.

In order to help place the potential risks associated with the OWMC Facility in context, the lifetime cancer risks corresponding to a variety of different exposures have been compiled and are presented in Table 12-1. As shown in the table, the cancer risks associated with exposures to routine atmospheric emissions from the proposed OWMC Facility are likely to be lower than many of those normally and routinely experienced. For example, the lifetime cancer risk resulting from an average medical diagnostic X-ray is estimated to be 1.4×10^{-3}, or between one and two in one thousand, whereas the greatest individual risk associated with any pathway for exposure to atmospheric emissions from the proposed OWMC Facility is likely to be less than 1×10^{-6}, or one in one million.

When making comparisons between the values presented in Table 12-1, it is important to note that different levels of uncertainty are associated with the various cancer risk estimates. For some of the estimates, e.g., cigarette smoking, the uncertainty is relatively small (Crouch and Wilson 1987). For others, such as drinking water with the USEPA limit for trichloroethylene, or exposure to emissions from the OWMC Facility, the uncertainty is higher. While it is believed that the approaches and assumptions used in the risk assessment are likely to overstate, rather than understate, the actual level of risk, the uncertainty associated with the evaluation of the proposed OWMC facility should be recognized in interpreting the results. Furthermore, the difference between "voluntary" and "involuntary" exposures should be considered when reviewing Table 12-1. Members of society are often willing to accept higher levels of risk for voluntary activities, such as smoking cigarettes, than for essentially involuntary exposures, such as those that may be associated with the operation of the proposed OWMC Facility.

To provide another frame of reference for evaluating the potential risks associated with the proposed OWMC Facility, the lifetime risks of death from various accidents have been compiled and are shown in Table 12-2. These risk estimates are all higher than the risks likely to be posed by any pathway of exposure to atmospheric emissions from the proposed facility.

TABLE 12-1

LIFETIME CANCER RISK ASSOCIATED WITH VARIOUS EXPOSURES[1]

Type of Exposure	Lifetime Risk	Source
• Average Medical Diagnostic X-Ray	1.4×10^{-3}	(1)
• 0.1 Kilogram Per Day of Beef with the Ontario Limit for Hexachlorobenzene	2.4×10^{-3}	(5)
• One Pint of Milk Per Day (aflatoxin)	1.4×10^{-4}	(1)
• One Half Pound of Charcoal Broiled Steak Per Week	2.1×10^{-5}	(1)
• Average Smoker (lifetime)	8.4×10^{-2}	(1)
• Sharing a Room With a Smoker (lifetime)	7.0×10^{-4}	(1)
• Living in a Home With the USEPA Standard for Radon	1.0×10^{-2}	(3)
• Drinking Water With the USEPA Limit for Trichloroethylene	1.4×10^{-7}	(2)
• Frequent Air Travel, 4 hrs/week (cosmic rays)	7.0×10^{-4}	(1)
• All Cancers, Ontario	2.6×10^{-1}	(4)
• Air Pollution, Eastern United States	1.4×10^{-2}	(2)
• Highest risk predicted for any pathway for exposure to routine emissions from the OWMC Facility	1.0×10^{-6}	

Sources:
(1) Crouch and Wilson, 1984
(2) Crouch and Wilson, 1987
(3) New York Times, May, 1986
(4) Statistics Canada 1984
(5) Derived from data provided by Ecologistics (1987)

[1] Except as noted, all statistics based on U.S. data. Where necessary, lifetime risks have been calculated by multiplying annual risks by 70 years.

TABLE 12-2

LIFETIME RISK OF DEATH ASSOCIATED WITH VARIOUS ACCIDENTS[1]

Type of Accident	Lifetime Risk	Source
• Lightning	3.1×10^{-5}	(1)
• Firearms (accidental)	7.1×10^{-4}	(1)
• Motor Vehicle	1.9×10^{-2}	(1)
• Electrocution	1.7×10^{-4}	(1)
• House Fires	2.1×10^{-3}	(1)
• All Farm Accidents	2.0×10^{-2}	(2)

Sources: (1) Statistics Canada 1984;
(2) Derived from data provided by the Farm Safety Association (1987) and the 1986 Census.

[1] All statistics based on Ontario data. Where necessary, lifetime risks have been calculated by multiplying annual risks by 70 years. (A factor of 50 years was used in converting annual farm accident related deaths to a lifetime basis.)

For example, the lifetime risk of death associated with farm accidents in the Province of Ontario is approximately 2 x 10^{-2}, or about one in fifty.

The nature of the risks associated with accidents are quite different from those resulting from exposure to carcinogens. Cancer may be produced as the result of continuous, long-term exposures; accidental deaths are caused by a single event. In addition, the development of cancer may not occur for many years after exposure; therefore, the incidence of cancer is generally higher for the elderly than it is for the young. Some types of accidents, on the other hand, are as common for children as they are for adults. Furthermore, the risks associated with accidents are typically known with greater certainty than those resulting from exposures to carcinogens. These factors should be recognized when making risk comparisons between accidental deaths and the probability of developing cancer.

Another measure by which the projected increase in cancer risk associated with the OWMC Facility can be judged is the current level of lifetime cancer risk in Canada and the Niagara Region. In 1981, approximately 25 percent of all deaths in Canada were attributed to cancer (Mills et al. 1986); similar cancer mortality rates have been found for Ontario and the Niagara Region (MOH 1984). This corresponds to an average lifetime cancer risk of about one in four for residents of Canada, Ontario, or the Niagara Region, as opposed to the predicted risk of approximately one in one million for the most significant pathway of exposure to atmospheric emissions from the OWMC Facility. Compared to this "background" level of cancer risk in the Niagara Region and throughout Canada, the potential risks posed by the OWMC Facility appear very small. Since the existing cancer rate is so high, however, some people believe that any additional risk, no matter how small, is unacceptable. It must be recognized, however, that there will be some risk associated with any type of waste treatment and disposal. The OWMC Facility will pose very low risks; efforts to reduce the existing cancer rate should not preclude the establishment of effective methods of addressing Ontario's need to develop additional waste disposal capacity.

Regulatory bodies and decision-makers often use the results of risk assessments in the development of standards and public policy. Many factors are considered in interpreting the results of an assessment, including the levels of predicted individual risk, the magnitude of the exposed population, and the uncertainty associated with the risk estimates. In the United States, cancer risks on the order of one-in-ten-thousand (1×10^{-4}) or lower have, in many cases, been judged to be insignificant (Travis et al. 1987). Thus, risks posed by the proposed OWMC facility fall well within the range that has been established as acceptable by regulatory bodies and decision-makers.

12.2.4 RECOMMENDATIONS

The human health risks associated with exposure to routine atmospheric emissions from the proposed OWMC Facility are low compared with those encountered in everyday life. Despite the comparatively low levels of risk associated with the routine emissions from the OWMC Facility, the protection of public health remains a key concern. Certain steps, therefore, should be taken to ensure the safe operation of the facility and to confirm that the health of nearby residents will not be jeopardized.

The assessment indicates that releases of certain metals, especially hexavalent chromium, and persistent organic chemicals, such as PCDD and PCDF, may pose the greatest potential for public health risk associated with incinerator stack emissions. Currently available stack gas sampling technologies do not permit the continuous monitoring of these contaminants. It is therefore recommended that a program of frequent periodic sampling and analysis for specific contaminants in the stack emissions be developed and implemented by OWMC. A similar program, focusing on organic chemicals, should also be developed for monitoring routine evaporator emissions. It is further recommended that sampling and analysis for volatile organic chemicals be performed on-site to ensure that potentially hazardous levels of fugitive emissions are not generated.

It should be noted that a series of monitoring programs have already been prepared by OWMC (Monenco 1987a). As the data from the analysis of incinerator stack, evaporator, and fugitive emissions becomes available, it is recommended that the results be incorporated into the risk assessment framework to determine the risks posed by actual operations.

As noted in the discussion of routine incinerator emissions, the bioaccumulation of environmentally persistent organics in food may contribute to total human exposures. It is therefore recommended that analysis of milk taken from dairy cows located near the area of maximum contamination be conducted to detect the presence of such chemicals as PCDD, PCDF, polychlorinated biphenyls (PCBs) and polynuclear aromatic hydrocarbons (PAH). The analysis of milk will provide an indication of the extent of any bioaccumulation of contaminants emitted from the OWMC Facility, and is less complex than the testing of beef, pork, or other animal tissues. Other types of biomonitoring, including the sampling and analysis of vegetation grown near the site, should also be undertaken. If the monitoring programs are to be initiated, the collection of data should begin at least one year (and preferably several years) prior to the start of facility operations to generate an understanding of background conditions.

The results of the environmental risk assessment suggest that adverse effects to plants and animals exposed to routine emissions from the OWMC facility are unlikely. It should be recognized, however, that environmental toxicity data are sparse for many plant and animal species. Thus, biomonitoring is recommended to confirm the results of the environmental risk assessment.

12.3. LEACHATE MIGRATION FROM THE ENGINEERED LANDFILL

12.3.1 HUMAN HEALTH RISK ASSESSMENT

The proposed OWMC Facility will include an engineered landfill for the disposal of solidified inorganic wastes. These wastes consist primarily of residues from the physical/chemical treatment plant, salt, ash, and slag from the incinerator, other inorganic materials that have been stabilized in on-site treatment units. Although these wastes are primarily inorganic, some may contain small quantities (less than 1 percent) of organics.

Leachate concentrations for various inorganics and organics that may be deposited in the OWMC engineered landfill were estimated using an approach outlined in Chapter 6 and described in detail in Appendix C. An illustrative analysis designed to provide an understanding of potential concentrations of organic chemicals in the ground water was also presented in Chapter 6.

In order to examine the potential risks associated with exposure to contaminants which may eventually reach the bedrock aquifer, estimated ground water concentrations can be compared to levels that could be considered "safe" for individual contaminants in drinking water. These levels, defined as "criterion" concentrations in this chapter, are presented in Table 12-3.

For non-carcinogens, criterion concentrations in drinking water were calculated using the following equation:

$$C_n = \frac{(ADI)(BW)}{I_w}$$

Where:

C_n = criterion concentration for a non-carcinogen in water, mg/L

ADI = Acceptable Daily Intake (ADI) value for the chemical, mg/(kg-d)

BW = body weight, 70 kg (NIOSH 1980)

I_w = water ingestion rate, 2 L/d (USEPA 1980)

TABLE 12-3

CRITERION CONCENTRATIONS

Contaminant	"Criterion" Concentration in Drinking Water[1] (mg/L)
Trichloroethylene	0.0032
Tetrachloroethylene	0.0007
Toluene	10.0
Xylene	36.0
Styrene	7.0
Cresol	1.8
Ethanol	1.7
Petroleum Distillates	3.5
PCDD/PCDF[2]	2.2×10^{-10}
Dichlorodifluoromethane	7.0
Trichlorotrifluoroethane	1050
Trichloroethane,1,1,1	1.0
Benzene	0.0012
Benzo(a)Anthracene	3.0×10^{-6}
Benzo(a)Pyrene	3.0×10^{-6}
Naphthalene	0.19
PCBs	8.1×10^{-6}
Formaldehyde	0.42

Continued . . .

TABLE 12-3 (continued)

CRITERION CONCENTRATIONS

Contaminant	"Criterion" Concentration in Drinking Water[1] (mg/L)
Phenol	3.5
Nitrate	35
Fluoride	2.1
Cyanide	0.7
Antimony	0.014
Arsenic	0.014
Barium	1.75
Cadmium	0.026
Chromium	0.18
Copper	1.00
Lead	0.14
Mercury	0.070
Nickel	0.53
Zinc	7.0

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[1] For non-carcinogens, "criterion" concentrations in drinking water are based on Acceptable Daily Intake (ADI) values. For carcinogens, "criterion" concentrations have been calculated using Unit Cancer Risk (UCR) values and a one-in-one million cancer risk level. In calculating the "criterion" concentration, it was assumed that the exposed individual is a 70 kg adult ingesting two liters of water per day.

[2] The criterion concentration for PCDD/PCDF is based on the congener distribution presented in Appendix C and the USEPA toxic equivalency scheme described in Appendix H.

Exposures to non-carcinogens present at levels in ground water that are lower than the criterion concentrations would not be expected to produce any adverse human health effects.

For carcinogens, criterion concentrations in drinking water were calculated as follows:

$$C_c = \frac{(1 \times 10^{-6})(BW)}{(UCR)(I_w)}$$

Where:

C_c = criterion concentration for carcinogen in water, mg/L

UCR = Unit Cancer Risk (UCR) value for the chemical, $[mg/(kg\text{-}d)]^{-1}$

BW = body weight, 70 kg (NIOSH 1980)

I_w = water ingestion rate, 2 L/d (USEPA 1980)

The use of the 1×10^{-6} factor in the above formula represents a level of risk; an individual whose entire lifetime drinking water supply is contaminated with the criterion concentration for the carcinogen would be predicted to have an increased lifetime cancer risk of one-in-one-million, or 1×10^{-6}. Other risk values, e.g., 1×10^{-5}, 1×10^{-7}, could also be used to develop criterion concentrations for carcinogens. For example, if a risk level of 1×10^{-5} were used, criterion concentrations would be 10 times higher than those calculated based on a 1×10^{-6} risk.

Estimated ground water concentrations and criterion concentrations for various metals are presented in Table 12-4.

TABLE 12-4

ESTIMATED CONCENTRATIONS OF METALS IN GROUND WATER

Contaminant	Estimated Ground Water Concentration[1] (mg/L)	"Criterion" Concentration[2] (mg/L)	OMOE Maximum Acceptable Concentration (MAC)[3] (mg/L)
Antimony	0.017	0.014	--
Arsenic	0.0033	0.014	0.05
Barium	0.10	1.75	--
Cadmium	0.0033	0.026	0.005
Chromium	0.0033	0.18	0.05
Copper	0.033	1.00	--
Lead	0.0033	0.14	0.05
Mercury	0.000033	0.070	0.001
Nickel	0.033	0.53	--
Zinc	0.033	7.0	--

---

[1] Based on leachate data provided by the Wastewater Technology Centre (Monenco 1987) and a three-fold dilution factor (Gartner Lee 1987). (Data for silver not available.)

[2] "Criterion" concentrations are based on Acceptable Daily Intake (ADI) values, and an assumed ingestion rate of two liters of water per day by a 70 kg adult.

[3] Source: OMOE 1987

These ground water concentrations reflect only the impact of dilution on the leachate; no other attenuation mechanisms are included. The data in Table 12-4 indicate that the migration of potentially hazardous metals from the OWMC landfill is not likely to pose a risk to public health. For most of the metals tested, predicted concentrations in the ground water are lower than the "criterion" concentration for drinking water. The only metal for which dilution alone would not result in an off-site concentration lower than the criterion concentration is antimony, for which the maximum ground water concentration is approximately equal to the criterion concentration.

It is also possible to compare the estimated ground water concentrations for some metals to Ontario Drinking Water Objectives established by the Ontario Ministry of the Environment (OMOE 1984). As shown on Table 12-4, estimated metal concentrations in ground water do not exceed any Maximum Acceptable Concentrations (MACs) set by OMOE.

Estimated levels of non-metals in leachate from the OWMC landfill are presented in Table 12-5, along with ground water concentrations generated in the illustrative analysis described in Chapter 6. In that analysis, degradation rates were assumed in order to provide an understanding of potential chemical concentrations in the bedrock aquifer underlying the preferred site.

TABLE 12-5

ESTIMATED CONCENTRATIONS OF NON-METALS IN LEACHATE AND GROUND WATER[1]

	Leachate Concentration (mg/L)	Ground Water Concentration[2] (mg/L) Case 1	Case 2
Low Molecular Weight Organics			
Trichloroethylene	4.7 E-02	1.5 E-08	1.0 E-03
Tetrachloroethylene	9.7 E-02	3.1 E-08	2.0 E-03
Toluene	8.6 E+00	2.7 E-06	1.8 E-01
Xylene	1.4 E-02	4.5 E-09	2.9 E-04
Styrene	9.0 E-03	2.8 E-09	1.9 E-04
Cresol	4.4 E-03	1.4 E-09	9.2 E-05
Ethanol	2.2 E+00	7.0 E-07	4.6 E-02
Petroleum Distillates	1.2 E-01	3.8 E-08	2.5 E-03
Dichlorodifluoromethane	5.8 E+00	1.8 E-06	1.2 E-01
Trichlorotrifluoroethane	1.2 E+01	3.7 E-06	2.4 E-01
Trichloroethane, 1,1,1	2.0 E-01	6.4 E-08	4.2 E-03
Benzene	1.7 E-02	5.4 E-09	3.6 E-04
Formaldehyde	6.9 E+01	2.2 E-05	1.4
Phenol	8.3 E-01	2.7 E-07	1.7 E-02
High Molecular Weight Organics			
Benzo(a)anthracene	1.6 E-04	5.1 E-11	3.3 E-06
Benzo(a)pyrene	7.0 E-05	2.2 E-11	1.5 E-06
PCDD/PCDF	7.7 E-09	2.4 E-15	1.6 E-10
PCBs	5.9 E-05	1.9 E-11	1.2 E-06
Naphthalene	3.2 E-03	1.0 E-09	6.7 E-05

TABLE 12-5 (continued)

ESTIMATED CONCENTRATIONS OF NON-METALS IN LEACHATE AND GROUND WATER[1]

	Leachate Concentration (mg/L)	Ground Water Concentration[2] (mg/L) Case 1	Case 2
Inorganics			
Nitrate	150	NC[3]	NC
Fluoride	1.8	NC	NC
Cyanide	3.6 E-04	NC	NC

---

[1] Leachate and ground water concentrations estimated using the approaches outlined in Chapter 6. Leachate concentration for PCDD/PCDF reported in 2,3,7,8 - TCDD equivalents based on the USEPA toxic equivalency scheme (see Appendix H). Leachate concentration for formaldehyde may be overestimated since actual loadings to the landfill may be in the form of a polymerized resin (Monenco 1987b).

[2] Case 1: Low molecular weight organics: Travel time to the aquifer assumed to be 1 000 years. Degradation half-life assumed to to 50 years.
High molecular weight organics: Travel time to the aquifer assumed to be 100 000 years. Degradation half-life assumed to be 5 000 years.

Case 2: Low molecular weight organics: Travel time to the aquifer assumed to be 1 000 years. Degradation half-life assumed to to 250 years.
High molecular weight organics: Travel time to the aquifer assumed to be 100 000 years. Degradation half-life assumed to be 25 000 years.

[3] NC: Not calculated.

As may be expected, leachate concentrations for many of the chemicals in Table 12-5 exceed the criterion concentrations listed in Table 12-3. However, if a 5 000-year degradation half-life is assumed for the high molecular weight organics, (benzo(a)anthracene, benzo(a)pyrene, PCDD/PCDF, PCBs, and naphthalene), and a 50-year half-life for the other organics, all ground water concentrations would be many times lower than the criterion concentrations. Even if much longer half-lives are assumed (25 000 years for high molecular weight organics, 250 years for the other organics) predicted ground water concentrations still do not exceed the criterion concentrations in most cases.

As previously noted, OMOE has developed drinking water objectives for several of the metals examined in the landfill assessment. Similar standards have not been developed for most of the organics evaluated. OMOE has, however, established an Interim Maximum Acceptable Concentration (IMAC) of 3×10^{-3} mg/L for PCBs (OMOE 1984). As shown in Table 12-4, the estimated concentration of PCBs in the leachate is approximately 6×10^{-5} mg/L. The predicted leachate concentration for PCBs is thus about 50 times lower than the IMAC set by OMOE.

Information compiled for OWMC by CANVIRO Consultants (1985) formed the basis for estimating the nature and concentration of trace organics in the wastes to be treated, solidified, and deposited in the OWMC landfill. Conservative assumptions were made using this information due to limitations in this data base. Furthermore, a separate analysis of potential loadings of organic contaminants to the landfill was conducted by the engineering consultants to OWMC (Monenco, 1987b). This assessment was based primarily on waste characterization data collected by the United States Environmental Protection Agency (USEPA 1983) as well as the information in the CANVIRO data base (1985). The results of the analysis conducted by the engineering consultants, which are included in Appendix M, also indicate that the risks associated with the operation of the proposed OWMC landfill are likely to be low.

12.3.2 RECOMMENDATIONS

Data predicting the human health risks that may be associated with landfill operations are sparse. Efforts have been made in the risk assessment to provide a conservative estimate of the quantities of potentially hazardous chemicals that may be present in leachate. It is recommended, however, that additional studies be undertaken to better characterize leachate quality and the fate and transport of chemicals released from the landfill.

For any landfill, including the one proposed by OWMC, it is appropriate to develop monitoring and contingency measures to ensure the protection of public health. Therefore, a monitoring program designed to provide the capability for early detection of the potential for significant ground water contamination should be adopted and implemented by OWMC. Based on the results of such monitoring, contingency measures, such as leachate collection or the institution of purge wells to collect ground water for treatment, could be implemented if necessary. It is also recommended that a specific protocol be adopted to identify and exclude from the landfill any wastes that may threaten to significantly degrade ground water quality.

12.4 ON-SITE UPSETS AND ACCIDENTS

12.4.1 HUMAN HEALTH RISK ASSESSMENT

OWMC is currently conducting a detailed hazard analysis of the proposed facility to identify on-site upset and accident scenarios that may pose significant risk to human health. Although the results of the analysis are not yet available, it has already been possible in the risk assessment to examine the potential off-site effects associated with several types of upsets and accidents similar to those that have occurred at existing hazardous waste treatment disposal facilities.

The potential human health risks associated with the on-site accidents and malfunctions evaluated were described in Section 10.4. The results of the assessment indicate the following:

- Chemical releases resulting from three scenarios examined (pH excursions during the treatment of cyanide wastes, pH excursions during metals precipitation using sulfide and the mixing of incompatible wastes) are not likely to lead to adverse effects in exposed individuals in the off-site community.
- The failure of the acid gas scrubbing system of the incinerator unit may cause minor, reversible effects, e.g., slight odors, temporary tightness of the chest, or irritation, in exposed individuals in the off-site community.
- Levels of off-site exposure resulting from a tank farm fire could be significant, although not likely to lead to incapacitating illness or death.

In evaluating the significance of any accident or upset, the frequency as well as the consequence must be considered. For example, the consequence of an airplane crash may well be death, but the frequency with which such an event occurs is sufficiently low so as not to preclude most people from flying. Recognizing the potentially serious consequences of a tank farm fire, a fault tree analysis was performed in Phase 4B to determine how often such an event might be expected to occur at the proposed OWMC Facility (Battelle 1987). As discussed in Appendix C, the results of the fault tree analysis indicate that a spill capable of leading to a fire would be expected to occur in the tank farm area approximately once every 4 years.

Based on the estimated ignition probability if a spill does take place, the frequency of a tank farm fire would likely not exceed one in 100 years. If individual halogenated waste tanks were to be segregated, the estimated frequency would drop to one in every 700 to 1 300 years. It should be recognized that certain measures that would mitigate a tank farm fire (e.g. fireproof insulation, segregation of waste tanks) were not included in the development of these estimates. Since some of these mitigation measures will be part of the final design, the frequency with which such an event could occur at the OWMC Facility will be significantly lower than calculated.

12.4.2 ENVIRONMENTAL RISK ASSESSMENT

Of the upset and accident events evaluated, only a tank farm fire appears to pose a significant environmental risk. The effects predicted in livestock and wildlife exposed to methylene chloride would be limited to temporary depression of the central nervous system. More severe irreversible effects would not be expected.

It should be noted that the chemicals examined in the assessment were selected, in part, on the basis of mammalian toxicity data. This selection process may have failed to identify chemicals of high acute toxicity to plants, fish, or birds. This limitation should be recognized in interpreting the results of the assessment.

12.4.3 RECOMMENDATIONS

The analysis of the tank farm fire has focused on the boil-off of volatile organic chemicals. It is possible, however, that a fire involving certain wastes may lead not to a boil-off, but rather to the generation of potentially hazardous chemicals as products of either complete or incomplete combustion. It is recommended that the public health risks associated with such an event be considered in the detailed hazard analysis currently underway.

OWMC will assist local municipal and regional authorities in the development of emergency response procedures to be followed in the event of an on-site upset or accident (Monenco 1987a). It is recommended that such plans be reviewed by OWMC following an evaluation of the results of the hazard analysis. The emergency procedures should address both on-site and off-site responses, such as community notification in the event of a potentially serious accident, and, if necessary, evacuation.

12.5 OFF-SITE TRANSPORTATION UPSETS AND ACCIDENTS

12.5.1 HUMAN HEALTH RISK ASSESSMENT

The public health risks associated with a special waste spill onto a roadway were discussed in Section 10.4.2. As indicated in that section, this spill scenario was predicted to have the potential to produce severe effects in exposed individuals.

The frequency with which a motor vehicle accident-related spill of special wastes onto a local access road might be expected is presented in Table 12-6. As indicated in the table, an accident-related spill of the most hazardous waste stream to be received at the OWMC Facility was predicted to occur approximately once every 1 320 years. Such a spill could result in significant levels of human exposure, although it would not be expected to produce fatalities. An accident-related spill which could result in some type of effect, ranging from minor irritation to a non-incapacitating illness, was estimated to occur approximately once in 132 years. An accident-related spill of any special waste onto the roadway is predicted to occur about once in 10.5 years. These frequencies do not include spills that might be caused by equipment failure, which are likely to be smaller than those associated with motor vehicle accidents.

As discussed in Section 10.4.2, ingestion of stream water contaminated by certain transportation spills could also result in potentially severe human health effects. Spills of chemicals which mix rapidly in the water column, such as pyridine, were predicted to be of particular concern.

TABLE 12-6

THE FREQUENCY OF TRUCK ACCIDENT-RELATED SPILLS OF SPECIAL WASTES ALONG THE WEST LINCOLN SITE ACCESS ROADS

(150,000 t/a Facility)

Type of Spill	Estimated Frequency
• Spill of any special waste	Once in 10.5 Years[1]
• Spill of a special waste that could result in some type of effect in exposed community	Once in 132 Years[2]
• Spill of the most hazardous special waste identified as to be received by the OWMC Facility	Once in 1 320 years[3]

---

[1] Based on the frequency for a truck accident-related spill (M.M. Dillon 1986b). This frequency applies to spills of any type of special waste. Spills of pumpable special waste were predicted to occur approximately once in fourteen years.

[2] Based on an estimate that spills of 8% of the special waste would result in adverse effects in the off-site population exposed to emissions resulting from a release onto the roadway (ENVIRON, 1985).

[3] Based on an estimate that only 0.8% of the special wastes are of the most hazardous composition examined in evaluating a spill onto the roadway (ENVIRON, 1985).

As noted in Chapter 8, however, spills of special waste into streams along the local access route are expected to have a very low probability of occurrence. For example, a spill of average size into Twenty Mile Creek, the largest stream along the local access route, would be expected to occur approximately once every 875 years. A large spill, in excess of 19 000 liters, would be predicted to occur approximately once in 8 750 years. Furthermore, the emergency response system planned by OWMC will permit rapid notification of such a spill to local residents, reducing the possibility that any individual would actually ingest the contaminated stream water.

12.5.2 ENVIRONMENTAL RISK ASSESSMENT

The results of the environmental risk assessment indicate that a spill of special wastes onto a roadway could pose some level of risk to certain environmental receptors. As discussed in section 11.5.3.7, the effects expected in exposed animals might include symptoms associated with temporary depression of the central nervous system, but are not predicted to include irreversible effects. The analysis indicated that crops and other plant life may not be as sensitive to short-term exposures to methylene chloride as are mammalian species.

The potential environmental impacts associated with a spill of special waste into a stream are discussed in Section 11.5.3.8. As with the human health risk assessment, severe potential environmental effects, such as fish kills, may be expected as the result of such a spill event. Again, the probability of such an event is very low

12.5.3 RECOMMENDATIONS

Despite the low frequency with which transportation spills are expected to occur, it is advisable to develop adequate emergency response protocols in light of the potential public health and environmental risks. For example, an exposure period of one hour was assumed in the analysis of the spill onto the roadway. The potential for adverse health effects would be reduced if rapid emergency response could limit exposures to a shorter period. Thus, appropriate placement of emergency equipment and personnel is recommended. It is also recommended that there be prompt remediation of spills which could result in potentially significant long-term exposures. An example would be the release [illegible] PCBs into Twenty Mile Creek or onto soil near the roadway.

It should be recognized that OWMC has made a commitment to assist local and regional authorities in preparing and implementing such emergency response plans (Monenco 1987a). This assistance will include: (1) training of response personnel; (2) selecting and acquiring of response equipment; and (3) providing technical advise regarding appropriate cleanup procedures in the event of an off-site release.

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GLOSSARY

Acceptable Daily Intake (ADI)	Estimate of the largest amount of chemical to which a person can be exposed on a daily basis without an adverse effect being anticipated (usually expressed in mg/kg/day).
Acute Exposure	Exposure of short duration. In this assessment, exposures of a few hours or less are termed acute.
Acute Safe Exposure Level (ASEL)	Limits applicable to predictable single exposures of short duration. If the ASEL is not exceeded, no effects in the exposed population would be anticipated.
Adjusted Margin of Safety (AMOS)	The largest amount of chemical to which a receptor can be exposed without an adverse effect being anticipated divided by the actual amount of exposure. An AMOS value greater than or equal to one indicates that an effect is not expected.
Adsorption	Adhesion of the molecules of a gas, liquid, or dissolved substance to a surface.
Atmospheric Dispersion	Dilution of gases or particles in the atmosphere.
Bioaccumulation	The process through which chemicals concentrate in plant and animal tissue.
Bioconcentration Factor	Ratio of the concentration of a substance in an organism to the concentration of the substance in an environmental medium (e.g. water, soil).
Biotransfer Factor	Measurement of the partitioning of a chemical from one medium to another.

GLOSSARY (continued)

Carcinogen	A substance that has the potential to produce cancer in an exposed population.
Chronic Exposure	Exposure of long duration. In this assessment, all continuous exposures occurring for a period of two weeks or longer are termed chronic.
Contaminant	A pollutant; an impurity.
Criterion Dose	Defined in the environmental risk assessment as a safe systemic dose.
Dose	Measurement of the amount of a chemical received by a receptor, per unit of body weight, per unit of time.
Dose-Response Relationship	The quantitative relationship between the level of exposure to a substance and the extent of toxic injury produced. In general, the frequency and/or severity of the effect is reduced as the level of exposure decreases.
Dry Deposition	The contact of very small particles and gases in the atmosphere with available surfaces as the result of turbulence and molecular motion.
Ecosystem	All organisms in a community and the associated environmental factors with which they interact.
Effluent	Liquid waste material discharged from an industrial process.
Environmental Persistence	The quality of being degraded slowly, if at all, in the environment.

GLOSSARY (continued)

Epidemiological Studies	Statistical evaluation of human populations to identify causes of disease. Such studies often compare the health status of a group of persons who have been exposed to a suspect agent with the health status of a comparable non-exposed group.
Exposure	To receive a chemical dose via ingestion, inhalation, or dermal contact.
Exposure Assessment	The component of risk assessment that involves an evaluation of the nature, magnitude, frequency, and duration of potential exposures.
Extrapolation	The estimation of a value beyond the range for which data are available.
Fault Tree Analysis	A process often used to evaluate the series of events which lead to an upset or accident.
Gestation	Period of pregnancy.
Hazard Analysis	The systematic identification of potential accidents and upset conditions at industrial facilities, and the quantitative analysis of their probability.
Hazard Evaluation	The component of risk assessment which involves gathering and evaluating data on the types of health injury or disease that may be produced by a chemical.
High-to-Low-Dose Extrapolation	The process of predicting low exposure risks from measured high exposure-high risk data.
Human Health Risk	The likelihood (or probability) that a given exposure or series of exposures to a toxic substance will damage the health of individuals experiencing the exposures.

Indicator Species	Those species specifically evaluated in the environmental risk assessment.
Ingestion	Type of exposure whereby chemicals enter the body through the mouth by consumption of solids or liquids.
Inhalation	Type of exposure whereby chemicals enter the body by being taken into the lungs.
Interspecies Extrapolation	Extrapolation of data from one species to another species for which data are unavailable.
Leachate	Aqueous liquid produced in a landfill by the infiltration of rain water or snowmelt.
Lifetime Exposure	Total amount of exposure to a substance that a human would receive in a lifetime (assumed to be seventy years in the current risk assessment).
Lowest-Observed-Adverse-Effect-Level (LOAEL)	Lowest dose level at which no adverse effects are noted in the exposed population.
Lowest-Observed-Effect-Level (LOEL)	Lowest dose level at which no effects are noted in the exposed population.
Margin-of-Safety (MOS)	Maximum amount of exposure producing no measurable effect in animals (or studied humans) divided by the actual amount of human exposure.
Mathematical Model	Representation of situations by mathematical formulae, e.g. numerical procedure used during risk assessment to perform extrapolations.

GLOSSARY (continued)

No-Observed-Adverse-Effect-Level (NOAEL)	Dose level at which no adverse effects are noted in the exposed population.
No-Observed-Effect-Level (NOEL)	Dose level at which no effects are noted in the exposed population.
Potable Water	Water suitable for drinking purposes.
Potency	A measure of the relative toxicity of a chemical.
Products of Incomplete Combustion (PICs)	Chemicals formed outside the combustion zone as a result of oxidation of chemicals due to the combination of molecular fragments.
Receptors	Members of a potentially exposed population.
Risk	Probability of injury, disease, or death under specific circumstances.
Risk Assessment	The process used to provide a quantitative estimate of the increased probability of injury, disease, or death under specific conditions of chemical exposure.
Risk Management	Decisions concerning (1) whether an assessed risk is sufficiently high to present a public health concern, and (2) the development of appropriate means for control of risks judged to be unacceptable.
Route of Exposure	Manner in which the chemical is introduced into the biological organism, e.g. inhalation, ingestion, skin absorption, etc.
Safe	Condition of exposure under which there is a "practical certainty" that no harm will result in exposed individuals.

GLOSSARY (continued)

Safety Factor	Adjustments to the experimentally observed NOEL, NOAEL, LOEL, or LOAEL values to compensate for uncertainties in the data and for imprecision in extrapolation to the general human population.
Sensitivity Analysis	Evaluation conducted to assess the impact of changes on the values of specific parameters.
Short Term Public Limit (STPL)	Limits applicable to predictable single exposures of short duration. If the STPL is not exceeded, the exposed population may be expected to exhibit effects which, at most, would be non-incapacitating and reversible.
Slag	The fused refuse produced by the solidification of a mixture of substances which have been heated to a temperature beyond their respective melting points.
Subchronic Exposure	Intermediate exposure period, generally ranging from approximately 2 days to 6 months.
Surrogate Chemicals	Chemicals chosen to represent the most hazardous waste constituents to be managed at the OWMC waste handling facility.
Systemic Dose	Dose received by the various organs and systems (e.g. reproductive, digestive) of the body as a result of absorption of a substance into the blood stream.
Systemic Effects	Effects observed at sites distant from the point of entry for a chemical as a result of its absorption into and distribution throughout the body.

GLOSSARY (continued)

Teratogen	A chemical or agent that causes physical defects in the developing embryo after maternal exposure to the chemical.
Threshold Dose	The dose that must be exceeded to produce a toxic response.
Toxic	Deleterious with respect to the effects produced by exposure to a chemical.
Toxic Equivalency Factor	Measure of the toxicity of one chemical substance relative to another.
Unit Cancer Risk (UCR)	Estimate of the incremental lifetime risk associated with each unit of exposure in the low exposure region.
Volatilization	Process of the release of vapor from the liquid phase.
Watershed	A region or area bounded by a divide as occupied by a drainage system.
Wet Deposition	The washout of contaminants from the atmosphere through precipitation.

Volume

2

Final report

OWMC
Ontario
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ENVIRON

Counsel in Health and Environmental Science

SITE ASSESSMENT
PHASE 4B: RISK ASSESSMENT
VOLUME 2
APPENDICES A THROUGH D

Prepared for

The Ontario Waste Management Corporation

Submitted by

ENVIRON Corporation

January, 1988

APPENDICES

A. WILDLIFE AND FISH SPECIES IN THE VICINITY OF THE PREFERRED SITE

B. CHARACTERIZATION OF WASTES

C. CHARACTERIZATION OF CONTAMINANT RELEASES

D. TRANSPORT AND FATE OF CONTAMINANTS RELEASED TO THE ATMOSPHERE

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APPENDIX A

WILDLIFE AND FISH SPECIES IN THE VICINITY OF THE PREFERRED SITE

The first step in the environment risk assessment for the proposed OWMC Facility was the identification of plants and animals that live in the vicinity of the preferred site. This appendix contains a description of:

- Wildlife reported in the vicinity of the proposed site; and
- Fish species in the Welland River and creeks near the site.

This information was used in the risk assessment to gain an understanding of the types of environmental species that may be impacted by emissions from the proposed OWMC Facility.

A.1 WILDLIFE

The preferred site and site vicinity (defined as an area 2 km outside the site boundary) serve as habitats for the region's naturally occurring wildlife. In general, the wildlife found on the site has been found to be similar to the wildlife in the site vicinity (Gartner Lee 1985).

As can be seen from Tables A-1, A-2, and A-3, a total of 107 bird species, 23 mammalian species, and 19 reptile and amphibian species have been identified on the site and in the site vicinity. Five species on these lists (eastern bluebird, orchard oriole, opossum, bobcat, and black rat snake) have been termed "rare" in terms of population size and distribtuion on a national, provincial, or regional level. These are not, however, considered endangered species.

A.2 FISH SPECIES

The fish species that have been reported in the Welland River and in the creeks near the preferred site are shown in Tables A-4 and A-5 (Gartner Lee 1986). The most abundant fish species collected in the Welland River were brown bullhead, white crappie, and sheepshead. The small intermittent creek that drains the preferred site (9C Creek) reportedly contains carp, central mudminnow,and brook stickleback. Common species in Beaver Creek included brown bullhead, northern pike, and bullfin. Although sampling was not conducted in Sucker Creek, the species of fish present in that stream are expected to be

TABLE A-1

BIRD SPECIES IDENTIFIED ON THE PREFERRED SITE OR IN THE SITE VICINITY

Common Name	Site	Vicinity (within a 2 km radius of the site)
Common Loon	X	
Great Blue Heron	X	X
Green-backed Heron		X
Black-crowned Night Heron		X
Wood Duck		X
Northern Pintail		X
Lesser Scaup		X
Sharp Shinned Hawk	X	
Coopers Hawk		X
Canada Goose		X
Mallard	X	X
Blue-winged Teal	X	X
Northern Harrier	X	
Turkey Vulture	X	X
Red-tailed Hawk	X	X
American Kestrel	X	X
Ring-necked Pheasant		X
Ruffed Grouse	X	X
Virginia Rail		X
Sora		X
Killdeer	X	X
Lesser Yellowlegs	X	X
Herring Gull	X	X
Yellow Bellied Sapsucker	X	X
Pileated Woodpecker		X
Spotted Sandpiper		X
Upland Sandpiper		X
Common Snipe	X	X
American Woodcock	X	X
Ring-billed Gull	X	
Rock Dove	X	X
Mourning Dove	X	X
Black-billed Cuckoo	X	X
Eastern Screech-Owl		X
Great Horned Owl	X	X
Chimney Swift	X	
Ruby-throated Hummingbird		X
Belted Kingfisher	X	
Red-headed Woodpecker	X	X
Downy Woodpecker	X	X
Hairy Woodpecker	X	
Northern Flicker	X	X

TABLE A-1 (continued)

BIRD SPECIES IDENTIFIED ON THE PREFERRED SITE OR IN THE SITE VICINITY

Common Name	Site	Vicinity (within a 2 km radius of the site)
Eastern Wood-Pewee	X	X
Alder Flycatcher	X	X
Willow Flycatcher	X	X
Least Flycatcher	X	X
Great Crested Flycatcher	X	X
Eastern Kingbird	X	X
Horned Lark	X	X
Purple Martin	X	X
Tree Swallow	X	X
Northern Rough-winged Swallow		X
Barn Swallow	X	X
Blue Jay	X	X
American Crow	X	X
Black-capped Chickadee	X	X
White-breasted Nuthatch	X	X
Brown Creeper	X	X
House Wren	X	X
Golden-crowned Kinglet	X	
Ruby-crowned Kinglet	X	
Yellow-throated Vireo		X
Blue-gray Gnatcatcher	X	X
Eastern Bluebird*	X	
Veery	X	X
Wood Thrush	X	X
American Robin	X	X
Gray Catbird	X	X
Northern Mockingbird	X	X
Brown Thrasher	X	X
Cedar Waxwing	X	X
European Starling	X	X
Warbling Vireo	X	X
Red-eyed Vireo	X	X
Yellow Warbler	X	X
Bay-breasted Warbler		X
Tennessee Warbler		X
Nashville Warbler		X
Chestnut-sided Warbler		X
Cap May Warbler		X
Yellow-rumped Warbler	X	X
Blackburnian Warbler		X
Cerulean Warbler	X	

TABLE A-1 (continued)

BIRD SPECIES IDENTIFIED ON THE PREFERRED SITE OR IN THE SITE VICINITY

Common Name	Site	Vicinity (within a 2 km radius of the site)
Black-throated Green Warbler	X	X
Ovenbird	X	X
Common Yellowthroat	X	X
Northern Cardinal	X	X
Rose-breasted Grosbeak	X	X
Indigo Bunting	X	X
American Redstart		X
Canada Warbler		X
Scarlet Tanager		X
Rufous-sided Towhee		X
American Tree Sparrow	X	
Chipping Sparrow	X	X
Field Sparrow	X	X
Vesper Sparrow	X	X
Savannah Sparrow	X	X
Song Sparrow	X	X
Swamp Sparrow	X	X
White-throated Sparrow	X	X
White-crowned Sparrow	X	
Dark-eyed Junco	X	X
Snow Bunting		X
Rusty Blackbird		X
Common Redpoli		X
Bobolink	X	X
Red-winged Blackbird	X	X
Eastern Meadowlark	X	X
Common Grackle	X	X
Brown-headed Cowbird	X	X
Orchard Oriole*	X	X
Northern Oriole	X	X
House Finch	X	
American Goldfinch	X	X
House Sparrow	X	X

---

* Denotes that the species is considered to be rare based upon the limited size and distribution of the species population on a national, provincial or regional level.

Source: Gartner Lee Associates Limited 1987.

TABLE A-2

MAMMALIAN SPECIES IDENTIFIED ON THE PREFERRED SITE OR IN THE SITE VICINITY

Common Name	Site	Vicinity (within a 2 km radius of the site)
Virginia Opossum*	X	X
Common Shrew	X	X
Short-tailed Shrew	X	X
Woodchuck		X
American Red Squirrel		X
Deer Mouse	X	
Eastern Cottontail	X	X
European Hare		X
Eastern Chipmunk		X
Grey Squirrel	X	X
White-footed Mouse	X	
Muskrat	X	X
Meadow Vole	X	
Norway Rat		X
Meadow Jumping Mouse	X	
Coyote	X	
Red Fox	X	X
Raccoon	X	X
American Mink	X	
Striped Skunk	X	
Lynx		X
Bobcat*	X	
White-tailed Deer	X	X

---

* Denotes that the species is considered to be rare based upon the limited size and distribution of the species population on a national, provincial or regional level.

Source: Gartner Lee Associates Limited 1987.

TABLE A-3

AMPHIBIAN AND REPTILE SPECIES IDENTIFIED ON THE PREFERRED SITE OR IN THE SITE VICINITY

Common Name	Site	Vicinity (within a 2 km radius of the site)
Eastern Newt		X
Redback Salamander	X	X
Stripped Chorus Frog	X	X
Green Frog	X	X
American Toad	X	X
Spring Peeper	X	X
Gray Treefrog	X	X
Western Chorus Frog	X	X
Bullfrog		X
Leopard Frog	X	X
Wood Frog	X	X
Snapping Turtle	X	X
Painted Turtle	X	X
Black Rat Snake*		X
Eastern Milk Snake		X
Garter Snake	X	X
Water Snake		X
Redbelly Snake	X	
Black Rat Snake	X	X

---

* Denotes that the species is considered to be rare based upon the limited size and distribution of the species population on a national, provincial or regional level.

Source: Gartner Lee Associates Limited 1987.

TABLE A-4

FISH SPECIES IDENTIFIED IN THE WELLAND RIVER, BEAVER CREEK AND/OR 9C CREEK

Common Name	Welland River	Beaver Creek	9C Creek
Bass, Rock	X		
Bass, White	X		
Bowfin	X	X	
Bullhead, Brown	X	X	
Carp	X	X	X
Catfish, Channel	X	X	
Crappie, Black	X		
Crappie, White	X	X	
Mudminnow, Central		X	X
Perch, White	X	X	
Perch, Yellow	X	X	
Pike Northern	X	X	
Pumpkinseed	X	X	
Redhorse, Shorthead	X	X	
Shad, Gizzard	X	X	
Sheepshead	X	X	
Shiner, Golden	X	X	
Stickleback, Brook		X	X
Sucker, White	X	X	

Source: Gartner Lee Associates Limited 1987.

TABLE A-5

FISH SPECIES IDENTIFIED IN TWENTY MILE CREEK, SIXTEEN MILE CREEK AND FIFTEEN MILE CREEK

Common Name	Twenty Mile Creek	Sixteen Mile Creek Mouth[a]	Fifteen Mile Creek Mouth[b]
Alewife		X	
Bass, Largemouth	X		
Bass, Rock	X		
Bullhead, Brown	X		
Crappie, Black	X		
Crappie, White	X		
Darter, Johnny	X		
Madtom, Tadpole	X		
Minnow, Bluntnose	X		
Minnow, Fathead	X		
Perch, Log	X		
Pike, Northern	X		
Pumpkinseed	X		
Salmon, Chinook	X	X	X
Salmon, Coho	X	X	X
Shiner, Golden	X		
Shiner, Sand	X		
Shiner, Spottail	X		
Sunfish, Green	X		
Trout, Brown	X	X	X
Trout, Rainbow	X	X	X

---

[a] From the mouth of Sixteen Mile Creek at Lake Ontario to the base of the Niagara Escarpment.

[b] From the mouth of Fifteen Mile Creek at Lake Ontario to the base of the Niagara Escarpment.

Source: Gartner Lee Associates Limited 1986.

similar to those in Beaver Creek. Several species of sport fish, including chinook salmon, coho salmon, rainbow trout,and brown trout, have been reported in the mouth of Fifteen, Sixteen and Twenty Mile Creeks.

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Gartner Lee Associates Limited. 1986. Fishery in Water Courses Surrounding Preferred Site LF-9C. Prepared for the Ontario Waste Management Corporation.

Gartner Lee Associates Limited. 1987. Site Assessment. Phase 4B: Biology. Final Draft Report. Prepared for the Ontario Waste Management Corporation.

APPENDIX B

CHARACTERIZATION OF WASTES

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APPENDIX B

CHARACTERIZATION OF WASTES

B.1 THE DATA BASE

B.1.1 INTRODUCTION

The wastes projected to be received at the OWMC facility can be classified according to the type of treatment expected:

- Organic Wastes, to be incinerated;
- Aqueous Organic Wastes, high water content streams to receive initial processing in the physical/chemical treatment plant;
- Inorganic Wastes, to be received by the physical/chemical treatment plant;
- Inorganic Wastes, to be sent directly to the solidification plant.

In order to characterize the contaminants found in these waste types, several sources of data have been used:

- OWMC Waste Profile;
- CANVIRO Data Base;
- Franklin Data Base.

This section of the appendix describes the sources of data used in the risk assessment to develop estimates of the quantities and concentrations of individual contaminants in each type of waste to be received at the OWMC facility. The general approach used to characterize the composition of the wastes using these data is also outlined.

B.1.2 THE OWMC WASTE PROFILE

As discussed in Chapter 4, OWMC and its engineering consultants have developed an estimate of the quantities and characteristics of the wastes to be received at the proposed special waste management facility. This profile, known as the OWMC Waste Profile, consists of 14 general classes of potentially hazardous wastes. The "typical" quantities of waste associated with each class as estimated in the OWMC Waste Profile are presented in Table B-1. The engineering consultants have further categorized the wastes listed in Table B-1 according to the type of treatment expected at the OWMC facility. The quantities of special waste associated with each treatment category are summarized in Table B-2.

The four general treatment categories of waste (organic, aqueous organic, and inorganic to either the physical/chemical treatment plant or the solidification plant) can be described in greater detail using a classification system developed at

TABLE B-1

THE OWMC WASTE PROFILE

	OWMC Waste Class	Typical Waste Quantity* (t/a)
1.	Organic Waste Sludges and Still Bottoms	11 050
2.	Solvents and Organic Solutions	3 330
3.	Oil and Grease	880
4.	Oil and Water Mixtures	3 535
5.	Organic and Oily Residuals	6 030
6.	Heavy Metal Solutions and Residuals	79 475
7.	Miscellaneous Chemicals and Products	3 100
8.	Paint and Organic Residuals	5 035
9.	Aqueous Solutions and Organics	1 785
10.	Anion Complexes	1 070
11.	Sludges and Inorganic Residuals	32 030
12.	Pesticides and Herbicide Waste	175
13.	PCB Wastes	35
14.	Cleanup Residuals	2 470
	TOTAL	150 000

---

*Rounded to the nearest 5 t/a.

SOURCE: OWMC Waste Profile (Monenco 1987).

TABLE B-2

THE TYPES OF WASTE PROJECTED

	Typical Waste Quantities (t/a)[2]			
OWMC Waste Class[1]	Organic Wastes	Aqueous Organic Wastes	Inorganic Wastes to the Physical/ Chemical Plant	Inorganic Wastes Sent Directly to the Solidification Plant
1	9 580	1 470	–	–
2	3 330	–	–	–
3	880	–	–	–
4	3 535	–	–	–
5	3 790	2 240	–	–
6	–	–	66 055	13 420
7	860	115	2 125	–
8	4 745	290	–	–
9	600	1 185	–	–
10	–	–	1 070	–
11	–	–	12 680	19 350
12	175	–	–	–
13	35	–	–	–
14	2 470	–	–	–
TOTAL	30 000	5 300	81 930	32 770

---

[1] Descriptions of the OWMC Waste Classes are provided in Table B-1.

[2] The total quantity of inorganic residues to be solidified will be increased by the transfer of treatment residuals. For example, residues from the physical/chemical treatment plant and ash from the incinerator will be sent to the solidification unit.

Source: OWMC Waste Profile (Monenco 1987) the University of California at Davis (UCD). In the UCD system, specific types of waste are grouped according to chemical composition and physical state. The UCD categories associated with the various wastes to be received at the OWMC facility are presented in Tables B-3 through B-6.

B.1.3 THE CANVIRO DATA BASE

A data base compiled by CANVIRO Consultants (1985) has been used in the risk assessment to define the identity and quantity of the individual constituents within each UCD category of waste listed in Tables B-3 through B-6. This data base was generated through a survey of 1 000 manufacturers in the Province of Ontario.

For the purposes of the risk assessment, the data compiled by CANVIRO is believed to provide the most appropriate basis for characterizing the composition of the special wastes to be received at the OWMC facility. As discussed in Chapter 4, however, there are a number of limitations associated with the use of the CANVIRO data base in the risk assessment. For example, contaminants in the data base are frequently described in only generic or descriptive terms, such as "aromatic hydrocarbon" or "adhesives", while specific chemical identities are necessary to evaluate rigorously the hazard associated with a given waste.

TABLE B-3

TYPICAL QUANTITIES OF ORGANIC WASTES

		Quantity (t/a)
OWMC CLASS 1: ORGANIC SLUDGE AND STILL BOTTOMS		
UCD		
247	Halogenated Still Bottoms	265
248	Non-halogenated Still Bottoms	1 060
252	Solid or Sludge Tannery Wastes	2 655
261	Polymeric Resin	2 650
263	Other Polymeric Wastes	1 180
446	Degreasing Sludge	885
523	Tank Bottom Sediments	885
	TOTAL	9 580
OWMC CLASS 2: SOLVENT AND ORGANIC SOLUTION		
UCD		
211	Halogenated Solvents with Heavy Metals	615
212	Halogenated Solvents with Other Metals	85
213	Other Halogenated Solvents	440
214	Non-halogenated Solvents with Heavy Metals	530
215	Non-halogenated Solvents with Other Metals	265
216	Other Non-halogenated Solvents	265
217	Unspecified Solvents	885
221	Organic Liquids with Metals and Halogens	45
222	Organic Liquids with Halogens Only	5
223	Organic Liquids with Metals Only	20
224	Other Organic Liquids	175
	TOTAL	3 330
OWMC CLASS 3: OIL AND GREASE		
UCD		
281	Waste Oil and Mixed Oil	880
OWMC CLASS 4: OIL AND WATER MIXTURES		
UCD		
283	Oily Mud, Sediment and Water	3 535

TABLE B-3 (continued)

TYPICAL QUANTITIES OF ORGANIC WASTES

		Quantity (t/a)
OWMC CLASS 5: ORGANIC AND OILY RESIDUES		
UCD		
264	Thermoset Wastes	10
282	Oily Tank Bottoms	395
284	Oily Acidic Solids	1 765
286	Oil/Water Separator Sludge	200
288	Oily Wastes with Heavy Metal Contaminants	885
289	Oily Wastes with Water	530
447	Tetraethyl Lead Sludge	5
	TOTAL	3 790
OWMC CLASS 7: MISCELLANEOUS CHEMICALS AND PRODUCTS		
UCD		
243	Pharmaceuticals and Wastes	180
245	Off-Spec or Aged Organics	440
416	Ink Sludge	60
513	Laboratory Waste Chemicals	180
	TOTAL	860
OWMC CLASS 8: PAINTS AND ORGANIC RESIDUES		
UCD		
254	Adhesives or Glue	445
258	Waste Tar and Residues	1 945
262	Latex Wastes	150
454	Paint Sludge	1 765
455	Paint Sludge Without Heavy Metals	440
	TOTAL	4 745
OWMC CLASS 9: AQUEOUS SOLUTION WITH ORGANICS		
UCD		
225	Aqueous Solution with Non-halogenated Residues Less Than 10%	140
226	Aqueous Solutions with Halogenated Residues Less Than 10%	10
227	Aqueous Solution with Non-halogenated Organic Residue Greater Than 10%	440
228	Aqueous Solution with Halogenated Organic Residue Greater Than 10%	10
	TOTAL	600

TABLE B-3 (continued)

TYPICAL QUANTITIES OF ORGANIC WASTES

UCD			Quantity (t/a)
OWMC CLASS 12: PESTICIDES AND HERBICIDES			
241	Pesticides and Wastes		130
532	Equipment and/or Containers Contaminated with Pesticides		45
		TOTAL	175
OWMC CLASS 13: PCB WASTES			
242	PCB Oils and High Strength (over 500 ppm) Liquids		35
OWMC CLASS 14: CLEAN UP RESIDUES			
512	Spill Cleanup		620
521	Spent Cartridge Filters		620
528	Pathogenic Wastes		615
531	Contaminated Rags		615
		TOTAL	2 470
TOTAL ORGANIC WASTES*			30 000

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* Does not include organics redirected to incinerator from the physical/chemical plant (approximately 530 t/a).

TABLE B-4

TYPICAL QUANTITIES OF AQUEOUS ORGANIC WASTES

		Quantity (t/a)
OWMC WASTE CLASS 1: ORGANIC SLUDGE AND STILL BOTTOMS		
UCD		
257	Phenolic Wastes	885
263	Other Polymeric Wastes	585
	TOTAL	1 470
OWMC WASTE CLASS 5: ORGANIC AND OILY RESIDUES		
UCD		
282	Oily Tank Bottoms	50
285	Oily Alkaline Solids	10
286	Oil/Water Separator Sludge	60
289	Oily Wastes with Water	2 120
	TOTAL	2 240
OWMC WASTE CLASS 7: MISCELLANEOUS CHEMICALS AND PRODUCTS		
UCD		
416	Ink Sludge	115
OWMC WASTE CLASS 8: PAINTS AND ORGANIC RESIDUALS		
UCD		
262	Latex Wastes	290
OWMC WASTE CLASS 9: AQUEOUS SOLUTION WITH ORGANICS		
UCD		
225	Aqueous Solution with Non-halogenated Residues less than 10%	1 185
TOTAL AQUEOUS ORGANIC WASTES		5 300

TABLE B-5

TYPICAL QUANTITIES OF INORGANIC WASTES TO BE RECEIVED AT THE PHYSICAL/CHEMICAL TREATMENT PLANT

		Quantity (t/a)
OWMC WASTE CLASS 6: HEAVY METAL SOLUTIONS AND RESIDUES		
UCD		
111	Acidic Solution with Heavy Metals	8 455
112	Acidic Solution with Other Metals and Non-metals	4 230
113	Other Acidic Solutions	3 170
121	Alkaline Solution with Heavy Metals	10 570
122	Alkaline Solution with Other Metals	530
123	Other Alkaline Solutions	3 910
131	Metal Finishing Solutions, Acid	1 585
132	Metal Finishing Solutions, Alkaline	530
133	Spent Pickle Liquor from Ferrous and Non-ferrous Metals	2 115
141	Aqueous Solutions with Heavy Metals	10 570
142	Aqueous Solutions with Other Metals	7 400
143	Aqueous Solutions with Reactive Anions	2 640
244	Photochemical Waste	530
251	Tannery Wastes - Liquid	2 115
431	Heavy Metal Sludge	3 805
433	Other Metal Sludge	2,115
445	Plating or Metal Finishing Sludge	740
523	Tank Bottom Sediments	1 055
	TOTAL	66 065
OWMC WASTE CLASS 7: MISCELLANEOUS CHEMICALS AND PRODUCTS		
UCD		
145	Brine	10
152	Inorganic Solid Chemicals	2 115
	TOTAL	2 125
OWMC WASTE CLASS 10: ANION COMPLEXES		
UCD		
146	Solutions Containing Cyanide	635
147	Solutions Containing Sulfides	10
444	Cyanide Bearing Sludge	425
	TOTAL	1 070
OWMC WASTE CLASS 11: SLUDGES AND INORGANIC RESIDUES		
UCD		
441	Lime Sludges	12 680
TOTAL INORGANIC WASTES TO BE SENT DIRECTLY TO THE PHYSICAL/CHEMICAL TREATMENT PLANT		81 940

TABLE B-6

TYPICAL QUANTITIES OF INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

		Quantity (t/a)
OWMC WASTE CLASS 6: HEAVY METAL SOLUTIONS AND RESIDUES		
UCD		
431	Heavy Metal Sludge	12 050
445	Plating or Metal Finishing Sludge	1 375
	TOTAL	13 425
OWMC WASTE CLASS 11: SLUDGES AND INORGANIC RESIDUES		
UCD		
151	Inorganic Solids	10 570
165	Spent Catalyst	210
167	Other Inorganic Waste	3 170
417	Alum and Gypsum Sludges	105
441	Lime Sludges	3 170
443	Sulfur Sludge	10
518	Dust Collector Wastes	2 115
	TOTAL	19 350
TOTAL INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT		32 775

To offset some of the limitations associated with the CANVIRO data base, ENVIRON has developed an approach for representing the entire quantity of potentially hazardous wastes by a number of specific "surrogate" chemicals. The application of this approach to surrogate selection is described in Section B.1.5.

B.1.4 THE FRANKLIN DATA BASE

The largest quantity of generically identified material in the CANVIRO data base is "oil and grease". The composition of this "oil and grease" cannot be estimated using the data provided by CANVIRO. For this reason, and since "oil and grease" represents a relatively large portion of the wastes to be received at the OWMC facility, it was deemed appropriate to characterize the composition of this material using an alternative data base developed by Franklin Associates (1985). The Franklin data base provides information on the approximate concentrations of a number of potentially hazardous chemicals that may be present in various types of used oils. In the risk assessment, data corresponding to "waste oil" was used to characterize the composition of the generic "oil and grease" identified in the CANVIRO data base. Waste oil is defined by Franklin Associates as "... a petroleum-derived oil which through use, storage, or handling has become unsuitable for its original purpose."

The Franklin data base includes a statistical summary of the concentrations of 18 potentially hazardous constituents in waste oil. These constituents are listed in Table B-7. The mean values for most constituents are greatly distorted by a few very high concentrations. It was therefore concluded by Franklin Associates that the median (50th percentile) is a better indicator of typical contaminant concentrations in waste oil than the mean. In the risk assessment, however, the 90th percentile concentrations, which are higher than the median concentrations, have been used to estimate conservatively the levels of 17 of the 18 chemicals in "oil and grease". The 75th percentile concentration was used for trichlorotrifluoroethane since data for this chemical were biased by a few very high values. For example, the highest concentration of trichlorotrifluoroethane reported in the Franklin data base was 550 000 ppm, or over 50 percent of the waste oil sample analyzed. It was judged that a stream with such a high fraction of a single specific chemical would not be classified as "oil and grease" in the CANVIRO data base.

Although the Franklin data base focuses on 18 frequently detected constituents of mixed and used oils, it is recognized that other hazardous chemicals may be present in this waste material. To assess the concentrations of these other chemicals, the United States Environmental Protection Agency (USEPA) commissioned a comprehensive analysis of 50 used oil

TABLE B-7

CONCENTRATION OF POTENTIALLY HAZARDOUS CONSTITUENTS IN WASTE OIL

	Concentration (ppm)		
	Median	75th Percentile	90th Percentile
Metals			
Arsenic	5	5	18
Barium	48	120	251
Cadmium	3	8	10
Chromium	6.5	12	35
Lead	240	740	1 200
Zinc	480	872	1 130
Organics			
Dichlorodifluoromethane	20	160	640
Trichlorotrifluoroethane	160	1 300	100 000
1,1,1-Trichloroethane	200	1 300	3 500
Trichloroethylene	100	200	800
Tetrachloroethylene	106	600	1 600
Benzene	20	110	300
Toluene	380	1 400	4 500
Xylene	550	1 400	3 200
Benzo(a)Anthracene	12	30	40
Benzo(a)Pyrene	10	12	16
Naphthalene	330	560	800
PCBs	5	15	50

SOURCE: Franklin Associates, 1985.

samples (Franklin 1985). In this investigation, the oil samples were analyzed for those organic chemicals designated as "priority pollutants" by the USEPA, as well as any other compounds detected at levels greater than 50 parts per million (ppm).

Based on the results of these analyses, ENVIRON has developed an estimate of the total concentration of those constituents in the generic "oil and grease" that may pose a significant risk to public health during handling and disposal at the OWMC facility. This total quantity was estimated as follows:

1. The average concentration for each organic "priority pollutant" detected in the oil samples was calculated. All "priority pollutants" not found in a sample were assumed to be present at the detection limit of the analysis (50 ppm).

2. The average concentration for each non-"priority pollutant" detected in the oil samples was calculated. Any non-"priority pollutant" found in one type of oil was assumed to be present at the detection limit (50 ppm) in those samples where it was not detected.

3. The total amount of potentially hazardous material in the "oil and grease" was estimated by summing the 75th

percentile concentration for trichlorotrifluoroethane, the 90th percentile concentrations for the other 17 frequently detected constituents (see Table B-7), the other "priority pollutants", and the non-"priority pollutants".

Based on this approach, it was estimated that approximately 3% of the "oil and grease" consists of potentially hazardous constituents. This value was used in calculating the total quantity of hazardous material associated with each treatment category of waste.

B.1.5 GENERAL APPROACH TO CHARACTERIZING THE COMPOSITION OF THE SPECIAL WASTES

As noted in the discussions of the CANVIRO and Franklin data bases in Chapter 4 and in the previous sections of this appendix, only a portion of the potentially hazardous constituents of the wastes to be received at the OWMC facility are sufficiently well-defined for the purposes of the risk assessment. Since the fraction of poorly defined (but possibly hazardous) material in these data bases is relatively large, an approach has been developed in the risk assessment to account for the total quantity of chemicals that may pose a significant risk to public health during the operation of the OWMC facility. This approach is based on the selection of well-defined toxic chemicals to act as "surrogates" in representing the total quantity of potentially hazardous material in the organic, aqueous organic, and inorganic wastes. The major components of the methodology used to estimate the quantity of "surrogate" chemicals in each type of waste are outlined below:

1. The OWMC Waste Profile was used to estimate the total quantity of each UCD category of waste to be received at the proposed facility.

2. The CANVIRO data base was used to determine the concentrations of various contaminants within each UCD category of waste.

3. The Franklin data base was used to estimate the composition of the "oil and grease" identified in the CANVIRO data base.

4. Each contaminant in the CANVIRO and Franklin data bases was classified as "hazardous" or "non-hazardous", based on its potential to pose a significant risk to public health during the operation of the proposed facility.

5. The total quantity of potentially "hazardous" waste was calculated.

6. The list of chemicals designated as potentially "hazardous" was reviewed to identify those which are sufficiently well-defined for the purposes of risk assessment. For some contaminants, such as "chlorinated ethylenes", judgments were made to estimate the composition in terms of specific chemicals.

7. "Surrogate" chemicals were selected from the list of sufficiently well-defined waste constituents. The total amount of "hazardous" waste was then assumed to be composed entirely of the relative quantities of the "surrogate" chemicals.

Details on the surrogate selection procedure for each treatment category of waste are provided in subsequent sections of this appendix.

B.2 ORGANIC WASTES

B.2.1 CHARACTERIZATION OF THE ORGANIC WASTES

B.2.1.1 Introduction

Approximately 30 000 t/a of primarily organic wastes are expected to be received at the proposed OWMC facility and treated in a rotary kiln incineration unit. To evaluate the potential human health risks associated with the releases from the incinerator stack, and fugitive emissions that may be generated during storage and handling, it is necessary to characterize the organic constituents of these wastes.

A portion of the metals present in the primarily organic wastes is also expected to be emitted from the incinerator stack. In the risk assessment, metal emissions from the OWMC incinerator have been predicted based on measurements recorded at existing hazardous waste incineration facilities. This approach was judged to be appropriate due to the relative lack of information on specific metals in the CANVIRO data base for organic wastes, and the availability of stack testing results from numerous hazardous waste incinerators. The metal content of the primarily organic wastes, therefore, has not been characterized using the CANVIRO data base.

B.2.1.2 Identification of Specific Hazardous Constituents of the Organic Wastes

Over 150 contaminants are identified in the CANVIRO data base as possible constituents of these organic wastes. The 50 contaminants predicted by the data base to be present in the largest quantities (excluding water) are those listed in Table B-8. A review of Table B-8 indicates that several

TABLE B-8

CHEMICALS PRESENT IN THE LARGEST QUANTITIES* (ORGANIC WASTES)

	Contaminant	Quantity Projected To Be Received at the OWMC Facility (t/a)
1.	Oil and Grease	3790
2.	Grit	2051
3.	Resins	1833
4.	Organic Solvents	990
5.	Tar/Asphalt	982
6.	Paint or Ink Pigments	932
7.	Leather	865
8.	Glass	822
9.	Leather Dust	749
10.	Cellulose	515
11.	Rubber	474
12.	Biological Matter	422
13.	Adhesives	404
14.	Iron	361
15.	Paper	358
16.	Metallic Oxides	352
17.	Polyurethane	347
18.	Mineral Spirits	319
19.	Ethylene Glycol	318
20.	Cotton	303
21.	Silica	298
22.	Plasticizers	284
23.	Alcohol	279
24.	Chlorinated Ethylenes	252
25.	Polyvinyl Chloride	221
26.	Toluene	212
27.	Solvents	198
28.	Polyethylene Waxes	195
29.	Varsol	192
30.	Urethane	187
31.	Latex	157
32.	Xylene	153
33.	Amines	145
34.	Organic Acids	144
35.	Soaps	132
36.	Aromatic Hydrocarbons	129
37.	Metals	129
38.	Chromium	125

TABLE B-8 (continued)

CHEMICALS PRESENT IN THE LARGEST QUANTITIES (ORGANIC WASTES)

	Contaminant	Quantity Projected To Be Received at the OWMC Facility (t/a)
39.	Calcium Carbonate	115
40.	Acetone	109
41.	Ketones	105
42.	1,1,1-Trichloroethane	100
43.	Methylene Chloride	96
44.	Bentonite/Clay	89
45.	Calcium Hydroxide	74
46.	Petroleum Waxes	70
47.	Calcium Oxide	64
48.	Polyethylene	63
49.	Caustic	61
50.	Vegetable Oil	60

---

* Excluding water (6 900 t/a)

Derived from the CANVIRO Data Base

contaminants, e.g. "oil and grease" and "chlorinated ethylenes", are described in only generic terms insufficient for the purposes of risk assessment. Given the large volume of "oil and grease" in the organic wastes, it was deemed appropriate to define the nature and quantity of specific hazardous constituents in this material. The composition of "oil and grease" has been characterized in this risk assessment using the Franklin data base, as shown in Table B-9. For other generically identified contaminants in the CANVIRO data base, assumptions with regard to chemical composition have been made where feasible. For example, "chlorinated ethylenes" was assumed to be a 50/50 mixture of trichloroethylene and tetrachloroethylene, the two most commonly occurring chlorinated ethylenes. "Aromatic hydrocarbons" was defined according to the relative quantities of specific aromatic hydrocarbons identified in the CANVIRO and Franklin data bases: benzene, naphthalene, benzo(a)pyrene, benzo(a)anthracene, chlorobenzenes, styrene, phenol, toluene and xylene. Similarly, "organic solvents" was assumed to be defined according to the relative quantities of the various solvents, e.g., benzene and toluene, identified in the CANVIRO and Franklin data bases.

From the information provided in the CANVIRO and Franklin data bases (and assumptions regarding the composition of certain generically defined contaminants), a list of

TABLE B-9

CHARACTERIZATION OF THE OIL AND GREASE IN THE ORGANIC WASTES

Contaminant	Concentration[1] (ppm)	Quantity[2] (t/a)
Dichlorodifluoromethane	640	2.4
Trichlorotrifluoroethane	1 300	4.9
1,1,1-Trichloroethane	3 500	13.3
Trichloroethylene	800	3.0
Tetrachloroethylene	1 600	6.1
Benzene	300	1.1
Toluene	4 500	17.1
Xylene	3 200	12.1
Benzo(a)Anthracene	40	0.15
Benzo(a)Pyrene	16	0.06
Naphthalene	800	3.0
PCBs	50	0.19

---

[1] Franklin Associates (1985). All values are 90th percentile concentrations except for the value reported for trichlorotrifluoroethane, which is a 75th percentile concentration.

[2] Based on 3 790 t/a "oil and grease".

contaminant quantities in the organic wastes was compiled. This list was screened to identify those organic chemicals for which sufficient toxicity data are currently available, and which may contribute to the risks associated with the operation of the OWMC facility. This screening process consisted of the elimination of metals, non-hazardous organics (such as vegetable oil), and inadequately defined substances (such as "plasticizers") from the list of organic waste constituents. The sufficiently well-defined hazardous chemicals thus selected are listed in Table B-10, along with quantities projected using the CANVIRO and Franklin data bases in conjunction with the OWMC Waste Profile.

B.2.1.3 Estimation of Total Hazardous Waste Quantity

As indicated above, variety of potentially hazardous liquid contaminants identified by CANVIRO in the organic wastes are too poorly defined to allow for any reasonable assumptions with regard to composition. Examples of such contaminants include "adhesives", "plasticizers", "amines" and "organic acids". In other cases, sufficient toxicity data are not available to evaluate the chronic health risks associated with exposures to certain chemicals (such as pentaerythritol) reported in the CANVIRO data base.

In the risk assessment, ENVIRON has taken account of those contaminants that are either poorly-defined or for which

TABLE B-10

HAZARDOUS CONSTITUENTS OF THE ORGANIC WASTES

Chemical	Quantity (t/a)
Acetone	312.7
Acrylonitrile	2.0
Benzene	22.5
Benzo(a)Anthracene	0.15
Benzo(a)Pyrene	0.061
Butadiene	0.18
Carbon tetrachloride	10.2
Chlorobenzene	0.13
Chloroform	17.0
Cresols	8.5
Dichlorobenzene	0.13
Dichlorodifluoromethane	2.4
Ethanol	472.6
Ethylene glycol	596.0
Ethylene oxide	0.15
Formaldehyde	5.4
Glycerine	2.2
Hexachlorobenzene	0.086
n-Hexane	107.2
Hydrazine	0.0005
Isopropyl alcohol	26.2
Kerosene	3.2
Methyl ethyl ketone	86.9
Methyl isobutyl ketone	33.9
Methylene chloride	167.1
Naphthalene	3.9
PCBs	21.6
Petroleum distillates	521.4
Phenol	57.0
Pyridine	10.0
Styrene	6.1
Tetrachloroethylene	253.1
Tetraethyl lead	0.040
Toluene	449.7
1,1,1-Trichloroethane	196.8
Trichloroethylene	224.6
Trichlorofluorocarbon	0.16
Trichlorotrifluoroethane	41.2
Xylene	228.4

All quantities derived from information in the CANVIRO (1985) and Franklin Associates (1985) data bases.

sufficient toxicity data are not available by conservatively classifying such chemicals as "hazardous liquids". Three classes of waste may then be defined: hazardous liquids, non-hazardous liquids, and solids. Using this approach, the following table was developed to describe the overall composition of the organic wastes:

Waste	Approximate Projected Quantity (t/a)
Hazardous Liquid	7 500
Non-hazardous Liquid	12 000
Solid	10 500
TOTAL	30 000

The greatest quantity of "non-hazardous liquid waste" is associated with water, (6 900 t/a) and the non-hazardous portion of oil and grease (3 700 t/a). The greatest quantity of "solid waste" is associated with grit (2 100 t/a), resins (1 800 t/a), leather and leather dust (1 600 t/a), and glass (800 t/a).

B.2.2 RANKING OF THE CHEMICALS PRESENT IN THE ORGANIC WASTES

B.2.2.1 Introduction

The list presented in Table B-10 represents a relatively long list of contaminants with considerable variation in both quantity and inherent hazard. For example, certain chemicals, e.g., trichlorofluorocarbon, are relatively non-toxic and are expected to be received at the facility in small quantities. Therefore, a methodology has been developed and implemented to identify those chemicals expected to be received in the largest amounts, and those which are likely to pose the greatest potential public health risk during operation of the proposed OWMC facility. This selection of a limited number of chemicals as indicators to evaluate the risks associated with a complex mixture is consistent with approaches adopted by various regulatory agencies (see for example, USEPA 1985). In this way, a manageable list of the chemicals of greatest concern have been chosen as "surrogates" to represent the entire quantity of "hazardous" liquid waste.

The criteria used to identify the chemicals of greatest concern in the organic wastes are (1) the toxicity of the constituent; (2) the quantity of chemical available for release (3) the potential for the constituent to be released to the environment. In addition to individual rankings on the basis of quantity and toxicity, separate rankings were performed based on the nature of the emissions. For example, the hazard associated with incinerator emissions can be related to the quantity and toxicity of the chemicals released. For fugitive emissions, the hazard has been based on three factors: toxicity, quantity and release potential.

The methodology for ranking the chemicals in Table B-10 according to these criteria is described in the following sections. The data used in the ranking process are shown in Tables B-11 and B-12.

B.2.2.2 Quantity

The first step in developing an estimate of the composition of the hazardous liquid portion of organic wastes was to rank the chemicals in Table B-10 according to quantity. The results of the ranking are presented in Table B-13. A review of Table B-13 indicates that ten chemicals compose approximately 90% of the liquid hazardous wastes projected to be incinerated at the OWMC facility.

B.2.2.3 Toxicity

A second ranking was conducted to determine which chemicals identified in the organic wastes to be received by OWMC are the most toxic. Since routine emissions are expected to involve the release of relatively small amounts of chemicals over a long period of time, chronic toxicity was used to rank the contaminants predicted by the CANVIRO data base to be present in the organic wastes.

As discussed in Chapter 9, chemicals can be classified as either carcinogens, which have the potential to cause cancer in

TABLE B-11

VAPOR PRESSURE DATA FOR HAZARDOUS CONSTITUENTS OF THE ORGANIC WASTES

Chemical	Vapor Pressure 20-25 C (mm Hg)	Vapor Pressure Reference
Acetone	2.34 E+02	Verschueren, 1983
Acrylonitrile	1.00 E+02	Verschueren, 1983
Benzene	9.45 E+01	Reid _et al._, 1977
Benzo(a)Anthracene	5.00 E-09	Mabey, 1982
Benzo(a)Pyrene	5.60 E-09	Mabey, 1982
Butadiene	2.10 E+03	Reid _et al._, 1977
Carbon tetrachloride	1.13 E+02	Verschueren, 1983
Chlorobenzene	1.20 E+01	Reid _et al._, 1977
Chloroform	1.93 E+02	Reid _et al._, 1977
Cresols	1.43 E-01	Reid _et al._, 1977
Dichlorobenzene	1.39 E+00	Reid _et al._, 1977
Dichlorodifluoromethane	4.25 E+03	Verschueren, 1983
Ethanol	5.86 E+01	Reid _et al._, 1977
Ethylene glycol	1.25 E-01	Verschueren, 1983
Ethylene oxide	1.10 E+03	Verschueren, 1983
Formaldehyde	3.83 E+03	Reid _et al._, 1977
Glycerine	1.37 E-05	Reid _et al._, 1977
Hexachlorobenzene	1.68 E-05	Mabey, 1982
n-Hexane	1.51 E+02	Reid _et al._, 1977
Hydrazine	1.60 E+01	Verschueren, 1983
Isopropyl alcohol	4.52 E+01	Reid _et al._, 1977
Kerosene	5.00 E+00	G.E., 1980
Methyl ethyl ketone	1.00 E+02	Reid _et al._, 1977
Methyl isobutyl ketone	1.96 E+01	Reid _et al._, 1977
Methylene chloride	4.33 E+02	Reid _et al._, 1977
Naphthalene	9.40 E-02	Mabey, 1982
PCBs	7.71 E-05	Mabey, 1982
Petroleum distillates	4.00 E+00	G.E., 1980
Phenol	5.00 E-01	Reid _et al._, 1977
Pyridine	2.06 E+01	Reid _et al._, 1977
Styrene	6.12 E+00	Reid _et al._, 1977
Tetrachloroethylene	1.40 E+01	Mabey, 1982
Tetraethyl lead	1.50 E-01	Verschueren, 1983
Toluene	2.82 E+01	Reid _et al._, 1977
1,1,1-Trichloroethane	1.00 E+02	Verschueren, 1983
Trichloroethylene	7.38 E+01	Reid _et al._, 1977
Trichlorofluorocarbon	6.67 E+02	Mabey, 1982
Trichlorotrifluoroethane	3.32 E+02	Verschueren, 1983
Xylene	5.80 E+00	Verschueren, 1983

TABLE B-12

TOXICITY DATA FOR HAZARDOUS CONSTITUENTS OF THE ORGANIC WASTES

Chemical	ADI (mg/kg/d)	UCR 1/(mg/kg/d)	ADI/UCR Reference
Acetone	2.86 E+00		USEPA, 1984
Acrylonitrile		2.40 E-01	CAG, 1985
Benzene		2.90 E-02	CAG, 1985
Benzo(a)Anthracene		1.15 E+01	Analogy to B(a)P
Benzo(a)Pyrene		1.15 E+01	CAG, 1985
Butadiene		1.80 E+00	CAG, 1985
Carbon tetrachloride		1.30 E-01	CAG, 1985
Chlorobenzene	3.00 E-02		USEPA, 1986
Chloroform		8.10 E-02	CAG, 1985
Cresols	5.00 E-02		USEPA, 1986
Dichlorobenzene	9.00 E-02		USEPA, 1986
Dichlorodifluoromethane	2.00 E-01		USEPA, 1986
Ethanol	4.95 E-02		ENVIRON, 1985
Ethylene glycol	1.00 E+00		ENVIRON, 1985
Ethylene oxide		3.50 E-01	CAG, 1985
Formaldehyde		1.12 E-02	ENVIRON, 1985
Glycerine	1.70 E-02		ENVIRON, 1985
Hexachlorobenzene		1.67 E+00	CAG, 1985
n-Hexane	5.21 E+00		ENVIRON, 1985
Hydrazine		3.90 E+00	CAG, 1985
Isopropyl alcohol	6.40 E-02		ENVIRON, 1985
Kerosene	2.40 E-02		ENVIRON, 1985
Methyl ethyl ketone	5.00 E-02		USEPA, 1986
Methyl isobutyl ketone	1.04 E-01		USEPA, 1984
Methylene chloride		1.40 E-02	CAG, 1985
Naphthalene	5.30 E-03		ENVIRON, 1985
PCBs		4.34 E+00	CAG, 1985
Petroleum distillates	1.00 E-01		ENVIRON, 1985
Phenol	1.00 E-01		USEPA, 1986
Pyridine	2.00 E-03		USEPA, 1986
Styrene	2.00 E-01		USEPA, 1986
Tetrachloroethylene		5.10 E-02	CAG, 1985
Tetraethyl lead	1.00 E-07		USEPA, 1986
Toluene	3.00 E-01		USEPA, 1986
1,1,1-Trichloroethane	2.90 E-02		Derived from McNutt *et al.* 1975
Trichloroethylene		1.10 E-02	CAG, 1985
Trichlorofluorocarbon	3.00 E-01		USEPA, 1986
Trichlorotrifluoroethane	3.00 E+01		USEPA, 1986
Xylene	1.03 E+00		ENVIRON, 1985

All quantities derived from information in the CANVIRO (1985) and Franklin Associates (1985) data bases.

TABLE B-13

RANKING ACCORDING TO QUANTITY

Chemical	Quantity (t/a)
Ethylene glycol	596.0
Petroleum distillates	521.4
Ethanol	472.6
Toluene	449.7
Acetone	312.7
Tetrachloroethylene	253.1
Xylene	228.4
Trichloroethylene	224.6
1,1,1-Trichloroethane	196.8
Methylene chloride	167.1
n-Hexane	107.2
Methyl ethyl ketone	86.9
Phenol	57.0
Trichlorotrifluoroethane	41.2
Methyl isobutyl ketone	33.9
Isopropyl alcohol	26.2
Benzene	22.5
PCBs	21.6
Chloroform	17.0
Carbon tetrachloride	10.2
Pyridine	10.0
Cresols	8.5
Styrene	6.1
Formaldehyde	5.4
Naphthalene	3.9
Kerosene	3.2
Dichlorodifluoromethane	2.4
Glycerine	2.2
Acrylonitrile	2.0
Butadiene	0.18
Trichlorofluorocarbon	0.16
Ethylene oxide	0.15
Benzo(a)Anthracene	0.15
Chlorobenzene	0.13
Dichlorobenzene	0.13
Hexachlorobenzene	0.09
Benzo(a)Pyrene	0.06
Tetraethyl lead	0.04
Hydrazine	0.001

Quantities derived from information in the CANVIRO (1985) and Franklin Associates (1985) data bases.

an exposed individual, or non-carcinogens, which do not. The potency of a carcinogen is usually characterized using a Unit Cancer Risk (UCR) value, whereas an Acceptable Daily Intake (ADI) value is the typical measure of chronic toxicity for a non-carcinogen. Since these different parameters are used to evaluate the risks associated with exposures to carcinogens and to non-carcinogens, an approach was needed for ranking both types of chemicals on the same scale. Such an approach was developed in conjunction with the Phase 4A generic risk assessment (ENVIRON 1985) and is discussed below.

Carcinogens are generally assumed to pose some level of risk at all finite exposure levels. The level of risk associated with an exposure to a carcinogen can be calculated using the following formula:

$$\text{Level of Risk} = (\text{Dose})(\text{UCR})$$

Although some risk is associated with any dose greater than zero, it is commonly accepted in regulatory decision-making that there is an exposure range below which the magnitude of cancer risk is very small. In many cases, lifetime cancer risks not exceeding a range of one-in-one hundred thousand to one-in-one-million (or 1×10^{-5} to 1×10^{-6}) have been considered negligible by various regulatory bodies (Rodricks et al. 1987).

ADI values that have been established for non-carcinogens are thought to reflect conditions of exposure that do not pose a risk to human health. It is not possible, however, to assert that a level corresponding to the ADI poses absolutely no risk to the exposed human population; at best, the risk is likely to be very small. A method of quantifying this "very small" level of risk is not currently available.

In the absence of evidence to the contrary and to provide a systematic means for comparing carcinogenic and non-carcinogenic compounds, it was assumed in the ranking of organic waste constituents that the "risk" associated with an exposure equivalent to an ADI is in the same range (10^{-5} to 10^{-6}) as that considered to be "very small" for carcinogens. This assumption provides a systematic means of comparing the risks associated with carcinogenic and non-carcinogenic compounds so as to allow the identification of the most toxic constituents of the organic wastes.

A methodology for calculating Risk Specific Dose (RSD) values for carcinogens and non-carcinogens based on the assumptions outlined above was developed in the Phase 4A generic risk assessment. RSD values, which allow for a ranking of the toxicity associated with all chemicals on the same scale, are calculated as follows:

- Non-carcinogens: $RSD = ADI$
- Carcinogens: $RSD = \frac{1 \times 10^{-6}}{UCR}$

Where:

RSD = Risk Specific Dose, mg/(kg-d)
ADI = Acceptable Daily Intake, mg/(kg-d)
UCR = Unit Cancer Risk, 1/[mg/(kg-d)]

The ranking of the chemicals identified in the organic wastes according to RSD values is presented in Table B-14. A review of the table reveals that, with the exception of tetraethyl lead, RSD values are significantly higher for carcinogens than for non-carcinogens.

B.2.2.4 Quantity and Toxicity

The hazard associated with incinerator emissions can be related to the quantity and toxicity of the chemicals released. Although it is known that some organics are destroyed more effectively via incineration than others (Dellinger et al. 1986), an accurate method for assessing "incinerability" of the wide range of chemicals expected at the OWMC facility is not currently available. The ranking of the constituents of the organic wastes according to the combination of quantity and toxicity has been based on the following formula:

$$QT = \frac{Qi}{RSDi}$$

Where:

QT = Quantity x toxicity ranking

TABLE B-14
RANKING ACCORDING TO TOXICITY

CHEMICAL	ADI (mg/kg/d)	UCR 1/(mg/kg/d)	RSD (mg/kg/d)
Benzo(a)Anthracene		1.15 E+01	8.70 E-08
Benzo(a)Pyrene		1.15 E+01	8.70 E-08
Tetraethyl lead	1.00 E-07		1.00 E-07
PCBs		4.34 E+00	2.30 E-07
Hydrazine		3.90 E+00	2.56 E-07
Butadiene		1.80 E+00	5.56 E-07
Hexachlorobenzene		1.67 E+00	5.99 E-07
Ethylene oxide		3.50 E-01	2.86 E-06
Acrylonitrile		2.40 E-01	4.17 E-06
Carbon tetrachloride		1.30 E-01	7.69 E-06
Chloroform		8.10 E-02	1.23 E-05
Tetrachloroethylene		5.10 E-02	1.96 E-05
Benzene		2.90 E-02	3.45 E-05
Methylene chloride		1.40 E-02	7.14 E-05
Formaldehyde		1.12 E-02	8.93 E-05
Trichloroethylene		1.10 E-02	9.09 E-05
Pyridine	2.00 E-03		2.00 E-03
Naphthalene	5.30 E-03		5.30 E-03
Glycerine	1.70 E-02		1.70 E-02
Kerosene	2.40 E-02		2.40 E-02
1,1,1-Trichloroethane	2.90 E-02		2.90 E-01
Chlorobenzene	3.00 E-02		3.00 E-02
Ethanol	4.95 E-02		4.95 E-02
Methyl ethyl ketone	5.00 E-02		5.00 E-02
Cresols	5.00 E-02		5.00 E-02
Isopropyl alcohol	6.40 E-02		6.40 E-02
Dichlorobenzene	9.00 E-02		9.00 E-02
Petroleum distillates	1.00 E-01		1.00 E-01
Phenol	1.00 E-01		1.00 E-01
Methyl isobutyl ketone	1.04 E-01		1.04 E-01
Styrene	2.00 E-01		2.00 E-01
Dichlorodifluoromethane	2.00 E-01		2.00 E-01
Toluene	3.00 E-01		3.00 E-01
Trichlorofluorocarbon	3.00 E-01		3.00 E-01
Ethylene glycol	1.00 E+00		1.00 E+00
Xylene	1.03 E+00		1.03 E+00
Acetone	2.86 E+00		2.86 E+00
n-Hexane	5.21 E+00		5.21 E+00
Trichlorotrifluoroethane	3.00 E+01		5.02 E+01

References for UCR and ADI values are provided in Table B-11.

Qi = Quantity of chemical i projected to be treated via incineration (t/a)

$RSDi$ = Risk Specific Dose for chemical i, mg/(kg-d)

The results of this ranking are presented in Table B-15. As with the ranking based on toxicity alone, the constituents with the highest scores are carcinogens. The most highly ranked non-carcinogen is tetraethyl lead, the 11th chemical on the list.

B.2.2.5 Quantity, Toxicity, and Vapor Pressure

The hazard associated with potential fugitive emissions has been based on three factors: 1) the toxicity of the constituent; 2) the quantity of the chemical available for release, and 3) the release potential associated with the constituent.

Based on an examination of the various formulas used to calculate fugitive emission rates in the risk assessment, vapor pressure was selected as the parameter to represent release potential. For example, losses from waste storage tanks are calculated using the equation:

$$Qi = (8.56 \times 10^{-2})(xi)(Pi)(Mi)$$

Where:

Qi = Emission rate for chemical i (g/s)

xi = Mole fraction of chemical i in the organic wastes, mol/mol

TABLE B-15

RANKING ACCORDING TO TOXICITY AND QUANTITY

CHEMICAL	Quantity (t/a)	RSD (mg/kg/d)	Q/RSD
PCBs	21.6	2.30 E-07	9.37 E+07
Tetrachloroethylene	253.1	1.96 E-05	1.29 E+07
Trichloroethylene	224.6	9.09 E-05	2.47 E+06
Methylene chloride	167.1	7.14 E-05	2.34 E+06
Benzo(a)Anthracene	0.015	8.70 E-08	1.74 E+06
Chloroform	17.0	1.23 E-05	1.38 E+06
Carbon tetrachloride	10.2	7.69 E-06	1.32 E+06
Benzo(a)Pyrene	0.061	8.70 E-08	6.98 E+05
Benzene	22.5	3.45 E-05	6.54 E+05
Acrylonitrile	2.0	4.17 E-06	4.83 E+05
Tetraethyl lead	0.04	1.00 E-07	4.00 E+05
Butadiene	0.18	5.56 E-07	3.21 E+05
Hexachlorobenzene	0.086	5.99 E-07	1.44 E+05
Formaldehyde	5.4	8.93 E-05	6.06 E+04
Ethylene oxide	0.15	2.86 E-06	5.36 E+04
Ethanol	472.6	4.95 E-02	9.55 E+03
1,1,1-Trichloroethane	196.8	2.90 E-02	6.79 E+03
Petroleum distillates	521.4	1.00 E-01	5.21 E+03
Pyridine	10.0	2.00 E-03	5.02 E+03
Hydrazine	0.0005	2.56 E-07	1.95 E+03
Methyl ethyl ketone	86.9	5.00 E-02	1.74 E+03
Toluene	449.7	3.00 E-01	1.50 E+03
Naphthalene	3.9	5.30 E-03	7.31 E+02
Ethylene glycol	596.0	1.00 E+00	5.96 E+02
Phenol	57.0	1.00 E-01	5.70 E+02
Isopropyl alcohol	26.2	6.40 E-02	4.10 E+02
Methyl isobutyl ketone	33.9	1.04 E-01	3.26 E+02
Xylene	228.4	1.03 E+00	2.22 E+02
Cresols	8.5	5.00 E-02	1.70 E+02
Kerosene	3.2	2.40 E-02	1.32 E+02
Glycerine	2.2	1.70 E-02	1.29 E+02
Acetone	312.7	2.86 E+00	1.09 E+02
Styrene	6.1	2.00 E-01	3.04 E+01
n-Hexane	107.2	5.21 E+00	2.06 E+01
Dichlorodifluoromethane	2.4	2.00 E-01	1.21 E+01
Chlorobenzene	0.13	3.00 E-02	4.38 E+00
Dichlorobenzene	0.13	9.00 E-02	1.46 E+00
Trichlorotrifluoroethane	41.2	3.00 E+01	1.37 E+00
Trichlorofluorocarbon	0.16	3.00 E-01	5.27 E-01

Quantities derived from information in the CANVIRO (1985) and Franklin Associates (1985) data bases. RSD values are taken from Table B-13.

P_i = Vapor pressure of chemical i at a temperature of approximately 25°C (mm Hg)

M_i = Molecular weight of chemical i (g/mol)

The product of the mole fraction (x_i) and the molecular weight (M_i) is related to quantity, which is a separate parameter in this ranking scheme. Inspection of the equation shows that vapor pressure is the most significant indicator of fugitive release potential given equal quantities of two substances.

Ranking the organic waste constituents according to the combination of quantity, toxicity, and vapor pressure has been based on the following formula:

$$QTP = \frac{(Q)(P)}{RSD}$$

Where:

Q = Quantity of chemical i projected to be present in the organic wastes (t/a)

P = Vapor pressure of chemical i at approximately 25 C, (mm Hg)

RSD = Risk Specific Dose for chemical i, mg/(kg-d)

The results of this ranking are presented in Table B-16. As with the rankings based on toxicity, and on the combination of toxicity and quantity, the constituents with the highest scores are, in general, carcinogens. The most highly ranked non-carcinogen is ethanol, the 11th chemical on the list.

TABLE B-16

RANKING OF ACCORDING TO QUANTITY, TOXICITY AND VAPOR PRESSURE

Chemical	Quantity (Q)(t/a)	RSD (mg/kg/d)	Vapor Pressure (P)(mm Hg)	(Q)(P)/RSD
Methylene chloride	167.1	7.14 E-05	4.33 E+02	1.01 E+09
Butadiene	0.18	5.56 E-07	2.10 E+03	6.75 E+08
Chloroform	17.0	1.23 E-05	1.93 E+02	2.66 E+08
Formaldehyde	5.4	8.93 E-05	3.83 E+03	2.32 E+08
Trichloroethylene	224.6	9.09 E-05	7.38 E+01	1.82 E+08
Tetrachloroethylene	253.1	1.96 E-05	1.40 E+01	1.81 E+08
Carbon tetrachloride	10.2	7.69 E-06	1.13 E+02	1.49 E+08
Benzene	22.5	3.45 E-05	9.45 E+01	6.18 E+07
Ethylene oxide	0.15	2.86 E-06	1.10 E+03	5.86 E+07
Acrylonitrile	2.0	4.17 E-06	1.00 E+02	4.80 E+07
1,1,1-Trichloroethane	196.8	2.90 E-02	1.00 E+02	6.79 E+05
Ethanol	472.6	4.95 E-02	5.86 E+01	5.60 E+05
Methyl ethyl ketone	86.9	5.00 E-02	1.00 E+02	1.74 E+05
Pyridine	10.0	2.00 E-03	2.06 E+01	1.03 E+05
Tetraethyl lead	0.04	1.00 E-07	1.50 E+02	6.00 E+04
Dichlorodifluoromethane	2.4	2.00 E-0	4.25 E+03	5.16 E+04
Toluene	449.7	3.00 E-01	2.82 E+01	4.23 E+04
Hydrazine	0.0005	2.56 E-07	1.60 E+01	3.12 E+04
Acetone	312.7	2.86 E+00	2.34 E+02	2.56 E+04
Petroleum distillates	521.4	1.00 E-01	4.00 E+00	2.09 E+04
Isopropyl alcohol	26.2	6.40 E-02	4.52 E+01	1.85 E+04
PCBs	21.6	2.30 E-07	7.71 E-05	7.22 E+03
Methyl isobutyl ketone	33.9	1.04 E-01	1.96 E+01	6.38 E+03
n-Hexane	107.2	5.21 E+00	1.51 E+02	3.11 E+03
Xylene	228.4	1.03 E+00	5.80 E+00	1.29 E+03
Kerosene	3.2	2.40 E-02	5.00 E+00	6.61 E+02
Trichlorotrifluoroethane	41.2	3.00 E-01	3.32 E+02	4.56 E+02
Trichlorofluorocarbon	0.16	3.00 E-01	6.67 E+02	3.51 E+02
Phenol	57.0	1.00 E-01	5.00 E-01	2.85 E+02
Styrene	6.1	2.00 E-01	6.12 E+00	1.86 E+02
Ethylene glycol	596.0	1.00 E+00	1.25 E-01	7.45 E+01
Naphthalene	3.9	5.30 E-03	9.40 E-02	6.92 E+01
Chlorobenzene	0.13	3.00 E-02	1.20 E+01	5.25 E+01
Cresols	8.5	5.00 E-02	1.43 E-01	2.43 E+01
Hexachlorobenzene	0.086	5.99 E-07	1.68 E-05	2.41 E+00
Dichlorobenzene	0.13	9.00 E-02	1.39 E+00	2.03 E+00
Benzo(a)Anthracene	0.15	8.70 E-08	5.00 E-09	8.62 E-03
Benzo(a)Pyrene	0.61	8.70 E-08	5.60 E-09	3.91 E-03
Glycerine	2.2	1.70 E-02	1.37 E-05	1.77 E-03

Quantities derived from information in the CANVIRO (1985) and Franklin Associates (1985) data bases. RSD values are taken from Table B-13.

B.2.3 SELECTION OF SURROGATE CHEMICALS

Based on the rankings presented in Tables B-13 through B-16, a group of 25 individual chemicals were selected to represent the "hazardous liquid" portion of the organic wastes. The steps in that selection process are outlined below.

Step 1

In order to explicitly include in the risk assessment those chemicals projected to constitute the greatest portion of the organic wastes, the following contaminants were selected as surrogates:

1. Ethylene Glycol
2. Petroleum Distillates
3. Ethanol
4. Toluene
5. Acetone
6. Tetrachloroethylene
7. Xylene
8. Trichloroethylene
9. 1,1,1-Trichloroethane
10. Methylene Chloride

These ten chemicals were identified in the ranking according to quantity (Table B-12) as accounting for approximately 90% of the well-defined hazardous waste material identified in the CANVIRO database.

Step 2

As previously noted in discussing the results of the ranking according to toxicity, and toxicity and quantity, carcinogens appear to pose the greatest potential risks associated with the operation of the proposed OWMC facility. This finding is consistent with the results of the Phase 4A generic risk assessment (ENVIRON 1985), which concluded that chronic exposures to non-carcinogens released from the facility are not expected to pose a risk to public health. Therefore, all carcinogens reported in the organic wastes have been selected as "surrogate" chemicals. It should be noted that certain carcinogens (tetrachloroethylene, trichloroethylene, and methylene chloride) were previously selected as surrogates strictly on the basis of projected quantities. The chemicals added to the list of surrogates as carcinogens were the following:

1. Polychlorinated biphenyls (PCBs)
2. Benzene
3. Chloroform
4. Carbon Tetrachloride
5. Formaldehyde
6. Acrylonitrile
7. Butadiene
8. Ethylene Oxide
9. Hexachlorobenzene
10. Benzo(a)pyrene
11. Benzo(a)anthracene
12. Hydrazine

Step 3

The third step in the selection of surrogate chemicals was to include those non-carcinogens that may pose a significant public health risk as constituents of incinerator emissions. This step was based on the ranking of chemicals according to the combination of toxicity and quantity and involved the following:

a) Calculation of the weighted average Q/RSD for the non-carcinogens previously selected as surrogates in Step 1.

b) Comparison of the weighted average Q/RSD value calculated in (a) with the individual Q/RSD values for each non-carcinogen not selected as a surrogate in Step 1.

The results of this comparison are summarized in Table B-17. As shown, two chemicals (pyridine and tetraethyl lead) were found to have Q/RSD values exceeding the weighted average Q/RSD for the previously selected non-carcinogens. Therefore, pyridine and tetraethyl lead were chosen as surrogates on the basis of the combination of toxicity and quantity.

Step 4

Finally, those non-carcinogens that may pose a significant public health risk as constituents of the fugitive emissions from the facility were selected as surrogates. These chemicals were identified as follows:

TABLE B-17

SELECTION OF SURROGATE CHEMICALS ON THE BASIS OF QUANTITY AND TOXICITY

Chemical	Q	Q/(RSD)	Weighted Q/(RSD)
Ethylene glycol	596	6.0 E+02	1.3 E+02
Petroleum distillates	521	5.2 E+03	9.8 E+02
Ethanol	473	9.5 E+03	1.6 E+03
Toluene	450	1.5 E+03	2.4 E+02
Acetone	313	1.1 E+02	1.2 E+01
Xylene	228	2.2 E+02	1.8 E+01
1,1,1-Trichloroethane	197	6.8 E+03	4.8 E+02
TOTAL	2,778		
Weighted average (Q)/RSD			3.5 E+03

Chemical	(Q)/RSD
Pyridine	5.0 E+03*
Tetraethyl lead	4.0 E+05*

---

* Selected as a surrogate on the basis of Q/(RSD). Values Q/(RSD) for all chemicals are shown in Table B-15.

a) Calculation of the weighted average (Q)(P)/RSD for the non-carcinogens previously selected as surrogates in Step 1.

b) Comparison of the weighted average (Q)(P)/RSD value calculated in (a) with the individual (Q)(P)/RSD values for each non-carcinogen not selected as a surrogate in Step 1.

The results of this comparison are summarized in Table B-18. Only one additional chemical, methyl ethyl ketone, was added to the list on the basis of this comparison.

B.2.4 ESTIMATION OF SURROGATE CHEMICAL QUANTITIES

The list of surrogates selected for the organic wastes is presented in Table B-19. The quantity associated with each surrogate has been calculated using the following formula:

$$Qsi = \frac{(Qi)(Qhl)}{Qt}$$

Where:

Qsi = Quantity of surrogate chemical, t/a;

Qi = Projected quantity of chemical to be received at the OWMC facility (as based on the CANVIRO and Franklin databases), t/a

Qhl = Projected quantity of "hazardous liquid" in the special wastes, 7 517 t/a

Qt = Total projected quantity of those chemicals selected as surrogates (as based on the CANVIRO and Franklin databases), 3 600 t/a

TABLE B-18

SELECTION OF SURROGATE CHEMICALS ON THE BASIS OF QUANTITY, TOXICITY, AND VAPOR PRESSURE

Chemical	Q	(Q)(P)/RSD	Weighted (Q)(P)/RSD
Ethylene glycol	596	7.5 E+01	1.6 E+01
Petroleum distillates	521	2.1 E+04	3.9 E+03
Ethanol	473	5.6 E+05	9.5 E+04
Toluene	450	4.2 E+04	6.8 E+03
Acetone	313	2.6 E+04	2.9 E+03
Xylene	228	1.3 E+03	1.1 E+02
1,1,1-Trichloroethane	197	6.8 E+05	4.8 E+04
TOTAL	2 778		
Weighted average (Q)/RSD			1.6 E+05

Chemical	(Q)(P)/RSD
Methyl ethyl ketone	1.7 E+05*

* Selected as a surrogate on the basis of (Q)(P)/RSD. Values (Q)(P)/RSD for all chemicals are shown in Table B-15.

TABLE B-19

MASS FRACTIONS OF SURROGATE CHEMICALS IN THE LIQUID PORTION OF THE ORGANIC WASTES

Chemical	Qi[1] (t/a)	Qsi[2] (t/a)	Msi[3] (t/t)
Acetone	312.7	653.2	3.4 E-02
Acrylonitrile	2.0	4.2	2.2 E-04
Benzene	22.5	47.1	2.4 E-03
Benzo(a)Anthracene	0.2	0.3	1.6 E-05
Benzo(a)Pyrene	0.1	0.1	6.5 E-06
Butadiene	0.2	0.4	1.9 E-05
Carbon tetrachloride	10.2	21.2	1.1 E-03
Chloroform	17.0	35.5	1.8 E-03
Ethanol	472.6	987.3	5.1 E-02
Ethylene glycol	596.0	1 244.9	6.4 E-02
Ethylene oxide	0.2	0.3	1.6 E-05
Formaldehyde	5.4	11.3	5.8 E-04
Hexachlorobenzene	0.1	0.2	9.2 E-06
Hydrazine	0.0	0.01	5.4 E-08
Methyl ethyl ketone	86.9	181.5	9.3 E-03
Methylene chloride	167.1	349.0	1.8 E-02
PCBs	21.6	45.1	2.3 E-03
Petroleum distillates	521.4	1 089.1	5.6 E-02
Pyridine	10.0	21.0	1.1 E-03
Tetrachloroethylene	253.1	528.6	2.7 E-02
Tetraethyl lead	0.0	0.1	4.3 E-06
Toluene	449.7	939.3	4.8 E-02
1,1,1-Trichloroethane	196.8	411.0	2.1 E-02
Trichloroethylene	224.6	469.1	2.4 E-02
Xylene	228.4	477.2	2.5 E-02
TOTAL	3 599	7 517	0.386

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[1] Projected quantity of chemical in the organic wastes to be received at the OWMC facility (as based on the CANVIRO and Franklin data bases.

[2] Quantity of surrogate chemical.

[3] Mass fraction of surrogate chemical in the liquid wastes.

Using this formula, the total quantity of "hazardous liquid" material in the organic waste is represented by the 25 surrogate chemicals.

The mass fraction of the surrogate chemicals in the liquid portion of the organic wastes have been calculated as follows:

$$Msi = \frac{Qsi}{(Qhl + Qnhl)}$$

Where:

Msi = Mass fraction of surrogate chemical i in the liquid wastes (t/t);

Qsi = Quantity of surrogate chemical i (t/a)

Qhl = Projected quantity of "hazardous" liquid in the special wastes, 7 517, t/a

Qnhl = Projected quantity of "non-hazardous" liquid in the special wastes, 12 000, t/a

The calculation of surrogate quantities and mass fractions in the liquid portion of the organic wastes is summarized in Table B-19.

B.3 AQUEOUS ORGANIC WASTES

B.3.1 CHARACTERIZATION OF THE AQUEOUS ORGANIC WASTES

Approximately 5 300 t/a of aqueous organic wastes are expected to be received by the proposed OWMC facility. Aqueous organic wastes are those streams that have high water content and are sent to the physical/chemical plant for initial processing. In the physical/chemical plant, organic material in the aqueous organic wastes will be separated from the water in emulsion breaking and dissolved air flotation (DAF) units and redirected to the incinerator. The remaining water will be polished using reverse osmosis and carbon adsorption so that it can be reused at the facility or discharged to an evaporation unit.

It is expected that some chemicals initially present in the aqueous organic wastes may ultimately be emitted from the evaporator. It is therefore necessary to characterize the concentrations of specific organic chemicals in the aqueous organic wastes to allow for an assessment of releases from the evaporator stack and as well as fugitive emissions from the DAF and emulsion breaking units.

The individual generator waste streams that would be treated in the OWMC aqueous organic system have been identified by the engineering consultants to OWMC (Monenco 1987). The general approach used in the risk assessment to select surrogate chemicals to represent these wastes is similar to that used for the organic wastes. First, a list of contaminants in the aqueous organic wastes was compiled. This list is shown in Table B-20

TABLE B-20

TYPICAL QUANTITIES OF SUBSTANCES OTHER THAN WATER IN THE AQUEOUS ORGANIC WASTES

Contaminant	Quantity (t/a)
Oil and grease	134.0
Sulfonic acid	48.2
Toluene	48.2
Phenols	19.4
Esters	11.5
Acetates	6.9
Ammonium hydroxide	4.8
Solvents	4.1
Azo/diazo dyes	2.7
Mineral spirits	1.9
Xylene	0.49
Formaldehyde	0.41
Surfactants	0.35
Varnish	0.25
Rust inhibitors	0.24
Organic solvents	0.12
Butadiene	0.11
Varsol	1.10
Bactericide/germicide	0.10
Acrylonitrile	1.08
Styrene	1.08
Kerosene	0.07
Fungicides	0.06
Mineral Acids	0.04
Amines	0.03
Sulfonates	0.002
Chlorinated paraffins	0.002
TOTAL	287.2

As shown in Table B-20, the contaminant present in the greatest quantity in aqueous organic waste is defined only as "oil and grease" in the CANVIRO data base. As discussed earlier in this appendix, the composition of "oil and grease" has been characterized using a data base developed by Franklin Associates (1985). Based on these data, the quantities of the specific hazardous organics in the "oil and grease" have been estimated and are shown in Table B-21.

In addition, contaminants referred to only as "solvents" and "organic solvents" have also been identified in the aqueous organic wastes using the CANVIRO data base. The only specific solvent identified in the aqueous organic streams is toluene. However, a small quantity of "chlorinated ethylenes" has been identified in certain wastes similar in nature to those classified as aqueous organic by the engineering consultants to OWMC. Therefore, the quantity of "solvents" and "organic solvents" in the aqueous organic wastes has been defined according to the relative quantities of three chemicals: toluene, tetrachloroethylene, and trichloroethylene.

As with the organic wastes, ENVIRON has taken account of those contaminants listed in Table B-21 which are either poorly defined (such as "acetates") or for which sufficient toxicity data are not available (such as sulfonic acid). This has been accomplished by classifying such contaminants as "hazardous".

TABLE B-21

CHARACTERIZATION OF THE OIL AND GREASE IN THE AQUEOUS ORGANIC WASTES

Contaminant	Concentration[1] (ppm)	Quantity[2] (t/a)
Dichlorodifluoromethane	640	0.086
Trichlorotrifluoroethane	1 300	0.17
1,1,1-Trichloroethane	3 500	0.47
Trichloroethylene	800	0.11
Tetrachloroethylene	1 600	0.21
Benzene	300	0.04
Toluene	4 500	0.60
Xylene	3 200	0.43
Benzo(a)Anthracene	40	0.005
Benzo(a)Pyrene	16	0.002
Naphthalene	800	0.11
PCBs	50	0.007

---

[1] Franklin Associates (1985). All values are 90th percentile concentrations except for the value reported for trichlorotrifluoroethane, which is a 75th percentile concentration.

[2] Based on 134.6 t/a "oil and grease".

Three classes of aqueous organic waste may then be defined: "hazardous", "non-hazardous" (other than water), and water. The non-hazardous waste other than water consists of that portion of the oil and grease which is not believed to pose a significant chronic health risk (see Section B.1.4). Using this approach, the following table was developed to describe the overall composition of the aqueous organic wastes:

Waste	Projected Approximate Quantity (t/a)
Hazardous	155
Non-hazardous (other than water)	130
Water	5 015

The estimation of the concentrations of the surrogate chemicals selected to represent the "hazardous" portion of the waste is described in the next section.

B.3.2 ESTIMATES OF SURROGATE CHEMICAL QUANTITIES

Characterization of the composition of "oil and grease", "solvents", and "organic solvents" and the elimination of inadequately defined contaminants yields a list of specific hazardous chemicals in the aqueous organic wastes. These chemicals, which are shown in Table B-22, have been used in the risk assessment as surrogates to represent the total quantity (155 t/a) of potentially hazardous substances defined as

TABLE B-22

MASS FRACTIONS OF SURROGATE CHEMICALS IN THE NON-WATER PORTION OF THE AQUEOUS ORGANIC WASTES

Chemical	Qi[1] (t/a)	Qsi[2] (t/a)	Msi[3] (t/t)
Toluene	53.0	102.7	3.6 E-01
Phenols	19.4	37.6	1.3 E-01
Petroleum distillates	3.0	5.8	2.0 E-02
Xylene	0.93	1.8	6.3 E-03
1,1,1-Trichloroethane	0.47	0.9	3.1 E-03
Styrene	1.08	2.1	7.3 E-03
Acrylonitrile	1.08	2.1	7.3 E-03
Formaldehyde	0.41	0.79	2.7 E-03
Tetrachloroethylene	0.24	0.47	1.6 E-03
Trichlorotrifluoroethane	0.17	0.33	1.1 E-03
Trichloroethylene	0.14	0.27	9.4 E-04
Naphthalene	0.11	0.21	7.3 E-04
Butadiene	0.11	0.21	7.3 E-04
Dichlorodifluoromethane	0.09	0.17	5.9 E-04
Benzene	0.04	0.08	2.8 E-04
PCBs	0.007	0.01	3.5 E-05
Benzo(a)Anthracene	0.005	0.01	3.5 E-05
Benzo(a)Pyrene	0.002	0.004	1.4 E-05
	80.3	155.6	0.54

---

[1] Projected quantity of chemical in the aqueous organic waste streams to be received at the OWMC facility (as based on the CANVIRO and Franklin data bases).

[2] Quantity of surrogate chemical.

[3] Mass fraction of surrogate chemical in the non-water portion of the aqueous organic wastes.

aqueous organic wastes. This has been accomplished using the following formula:

$$Qsi = \frac{(Qi)(Qhl)}{Qt}$$

Where:

Qsi = Quantity of surrogate chemical, t/a

Qi = Projected quantity of chemical to be received at the OWMC facility (as estimated using the CANVIRO and Franklin data bases), t/a

Qhl= Projected quantity of "hazardous" material in the aqueous organic wastes, 155 t/a

Qt = Total projected quantity of those chemicals selected as surrogates (estimated using the CANVIRO and Franklin data bases), 80 t/a

The mass fractions for the surrogate chemicals in the non-water portion of these wastes has been calculated as:

$$Msi = \frac{Qsi}{Qnw}$$

Where:

Msi = Mass fraction of surrogate chemical i in the non-water wastes (t/t)

Qsi= Quantity of surrogate chemical i (t/a)

Qnw = Projected quantity of material other than water in the aqueous organic wastes, 285 t/a

Water was not included in calculating the contaminant mass fractions in the aqueous organic material since the organic constituents may form a separate phase in the physical/chemical plant, thereby leading to increased chemical concentrations in that separate phase. The chemical mass fractions in this organic phase are used in the estimation of fugitive emissions from the dissolved air flotation (DAF) and emulsion breaking units of the physical/chemical plant. The calculation of the surrogate chemical quantities and mass fractions in the organic component of the aqueous wastes is summarized in Table B-22.

B.4 INORGANIC WASTES

B.4.1 CHARACTERIZATION OF THE INORGANIC WASTES

Approximately 115 000 t/a of primarily inorganic wastes are projected to be received by the proposed OWMC facility (Monenco 1987). Of these wastes, approximately 82 000 t/a are to be treated in the physical/chemical plant, and 33 000 t/a are to be sent directly to the solidification unit. It should be noted that relatively large quantities of solid residues from both the incinerator and the physical/chemical treatment plant will also be stabilized in the solidification unit prior to placement in the engineered landfill.

Liquid effluent remaining after the treatment of the inorganic wastes in the physical chemical plant will be discharged to an evaporation unit. This effluent will consist primarily of water, but may be contaminated with both organics and metals originally present in the inorganic wastes sent to the physical/chemical treatment plant. Non-pumpable residues generated as a by-product of the treatment processes will be stabilized along with the inorganic wastes sent directly to the solidification plant prior to disposal in the engineered landfill. Therefore, metals and organics from the inorganic wastes received by the physical/chemical treatment plant may be emitted from the evaporator, and contaminants in the wastes sent to solidification may be expected in leachate generated in the landfill.

The contaminants expected to be present in the largest quantities in the inorganic wastes are listed in Table B-23 and B-24. The quantities in the two tables are based on information provided in the OWMC Waste Profile (Monenco 1987), and the CANVIRO data base (CANVIRO 1985). As shown in Table B-23, the inorganic wastes to be sent to the physical/chemical treatment plant are expected to contain large quantities of alkaline and acid substances. Metals, and to a much lesser degree organics, will also be present in these wastes. The inorganic wastes to be sent directly to solidification are expected to be composed primarily of silica, carbonates, and various metals.

In developing an estimate of the composition of the inorganic wastes for the purposes of the risk assessment, the analysis has been limited to those constituents that are

TABLE B-23

TYPICAL CONTAMINANT QUANTITIES IN THE INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Contaminant	Quantity (t/a)[1]
Mineral Acids	2 502
Sulphuric Acid	1 488
Calcium Hydroxide	1 425
Calcium Carbonate	1 277
Caustic	559
Iron	468
Aluminum	459
Ammonium Phosphate	448
Zinc	416
Sulphur	400
Anhydrides	380
Ammonium Nitrate	380
Urea	380
Calcium Sulphate	376
Phosphate	349
Sodium Salts	335
Acids	267
Sulfonic Acid	238
Cellulose	235
Sodium Chloride	218
Ammonium Chloride	207
Nitric Acid	116
Copper	85
Chromium	82
Nickel	74

---

1 Quantities based on Monenco (1987), and CANVIRO (1985).

TABLE B-24

TYPICAL CONTAMINANT QUANTITIES IN THE INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Contaminant	Quantity (t/a)[1]
Silica	8 520
Calcium Carbonate	3 147
Iron	1 377
Metals	968
Metal Chips/Dust	793
Aluminum Oxide	737
Sulphur	699
Zinc	641
Copper	470
Lead	428
Calcium Hydroxide	362
Calcium Oxide	359
Zinc Oxide	268
Bentonite/Clay	215
Aluminum	191
Chromium	156
Phosphate	156
Sodium Chloride	129
Oil and Grease	104
Nickel	98
Calcium sulphate	95
Zirconium	87
Phenols	76
Cellulose	70
Ferric Oxide	64

---

[1] Quantities based on Monenco (1987), and CANVIRO (1985).

believed to have the potential to pose a significant risk to public health during handling at the proposed facility. Contaminants such as grit and potassium, which are generally believed to be relatively non-toxic, have been considered "non-hazardous". In addition, compounds such as sulfuric acid that would be expected to be neutralized in the physical/chemical treatment plant or in the solidification unit were not considered in the risk assessment. The contaminants used to estimate the quantity and composition of potentially "hazardous" material in the inorganic wastes are listed in Tables B-25 and B-26.

As with the organic and aqueous organic streams, the composition of "oil and grease" reported in the CANVIRO data base has been characterized using information derived from a study conducted by Franklin Associates (Franklin Associates 1985). The well-defined toxic constituents of "oil and grease" in the inorganic wastes are presented in Tables B-27 and B-28.

Sufficient toxicological data are not currently available for several chemicals (e.g., furfural, aluminum) listed in Tables B-25 and B-26. In the analysis of the inorganic wastes, the chemicals for which sufficient toxicity data are not available have been separated into two categories of potentially hazardous materials: metals and organics. In addition, substances that may be toxic but that are poorly

TABLE B-25

TYPICAL QUANTITIES OF POTENTIALLY HAZARDOUS SUBSTANCES IN THE INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Contaminant	Quantity (t/a)[1]
Aluminum	459.0
Zinc	416.1
Anhydrides	379.8
Ammonium nitrate	See note [2]
Copper	84.9
Chromium	81.9
Nickel	73.9
Potassium nitrate	See note [2]
Toluene	66.1
Oil and grease	49.2
Paint or ink pigments	45.8
Cobalt	26.0
Cyanide	22.9
Heavy metal hydroxides	20.8
Developer solution	20.6
Phenols	20.2
Chromic acid	17.0
Tin	11.3
Silver	9.7
Photochemicals	9.7
Vanadium	8.8
Organic acids	8.7
Cadmium	8.5
Tin compounds	8.4
Styrene	7.9
Acrylonitrile	7.9
Metals	7.8
Chromates	6.9
Silver bromide	6.7
Nickel hydroxide	6.4
Aluminum oxide	5.8
Lead	5.5
Photographic fixing	5.0
Arsenic	3.7
Antimony	3.7
Aluminum chloride	3.1
Solvents	2.1
Nitrate	1.6
Lead oxide	1.3
Chromium salts	1.0

TABLE B-25 (continued)

TYPICAL QUANTITIES OF POTENTIALLY HAZARDOUS SUBSTANCES IN THE INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Contaminant	Quantity (t/a)[1]
Boron	0.84
Mineral spirits	0.84
Sodium aluminate	0.62
Organic chemicals	0.51
Azo/diazo dyes	0.5
Mercury	0.43
Cresols	0.40
Ethylene glycol	0.36
Organic solvents	0.17
Palladium	0.14
Ammonium bifluoride	0.061
Zinc sulfate	0.045
Sodium fluoride	0.009
Lead Hydroxide	0.002
Lead chloride	0.002
TOTAL	2 310.0

---

1 Quantities based on Monenco (1987) and CANVIRO (1985)

2 According to the CANVIRO data base, approximately 380 t/a of ammonium nitrate and 70 t/a of potassium nitrate would be predicted to be sent to the physical/chemical treatment plant. The engineering consultants to OWMC, however, have determined that it is unlikely that substantial quantities of nitrate, which is a valuable commodity, would actually be received.

TABLE B-26

TYPICAL QUANTITIES OF POTENTIALLY HAZARDOUS SUBSTANCES IN THE INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Contaminant	Quantity (t/a)[1]
Metals	968.1
Aluminum oxide	736.7
Zinc	640.7
Copper	469.9
Lead	427.7
Zinc oxide	267.8
Aluminum	190.8
Chromium	156.1
Oil and grease	103.7
Nickel	97.7
Phenols	75.8
Heavy metal hydroxides	63.4
Lead oxide	47.5
Formaldehyde	42.7
Toluene	40.0
Furfural	39.8
Vanadium	28.7
Nickel hydroxide	20.2
Benzothiazoles	16.2
Tin	15.8
Tin compounds	7.3
Nitrate	4.9
Metallic oxides	4.5
Organic acids	3.6
Organic solvents	3.0
Oxalic acid	2.9
Mineral spirits	2.7
Cadmium	2.1
Sodium aluminate	2.0
Organic chemicals	1.6
Mercury	1.4
Cyanates	1.0
Triethyl aluminum	0.72
Xylene	0.51
Silver	0.51
Chromates	0.38
Paint or ink pigments	0.38
Palladium	0.25
Cobalt	0.25
Chromium alkyl aluminum	0.14

TABLE B-26 (continued)

TYPICAL QUANTITIES OF POTENTIALLY HAZARDOUS SUBSTANCES IN THE INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Contaminant	Quantity (t/a)[1]
Azo/diazo dyes	0.13
Chromic acid	0.12
Aromatic hydrocarbons	0.11
Cresols	0.10
Alcohol	0.073
Chlorinated hydrocarbons	0.045
Arsenic	0.040
Nickel chloride	0.020
Acetates	0.020
Fluoride	0.014
Chromium salts	0.010
Lead chloride	0.004
Lead hydroxide	0.004
TOTAL	4 490.0

---

[1] Quantities based on Monenco (1987) and CANVIRO (1985)

TABLE B-27

CHARACTERIZATION OF THE OIL AND GREASE IN THE INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Contaminant	Concentration[1] (ppm)	Quantity[2] (t/a)
Dichlorodifluoromethane	640	0.03
Trichlorotrifluoroethane	1 300	0.06
1,1,1-Trichloroethane	3 500	0.17
Trichloroethylene	800	0.04
Tetrachloroethylene	1 600	0.08
Benzene	300	0.01
Toluene	4 500	0.22
Xylene	3 200	0.16
Benzo(a)Anthracene	40	0.002
Benzo(a)Pyrene	16	0.001
Naphthalene	800	0.04
PCBs	50	0.002
Arsenic	18	0.001
Barium	251	0.012
Cadmium	10	0.0005
Chromium	35	0.002
Lead	1 200	0.059
Zinc	1 130	0.056

---

[1] Franklin Associates (1985). All values are 90th percentile concentrations except for the value reported for trichlorotrifluoroethane, which is a 75th percentile concentration.

[2] Based on 49.2 t/a "oil and grease".

TABLE B-28

CHARACTERIZATION OF THE OIL AND GREASE IN THE INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Contaminant	Concentration[1] (ppm)	Quantity[2] (t/a)
Dichlorodifluoromethane	640	0.07
Trichlorotrifluoroethane	1 300	0.13
1,1,1-Trichloroethane	3 500	0.36
Trichloroethylene	800	0.08
Tetrachloroethylene	1 600	0.17
Benzene	300	0.03
Toluene	4 500	0.47
Xylene	3 200	0.33
Benzo(a)Anthracene	40	0.004
Benzo(a)Pyrene	16	0.002
Naphthalene	800	0.08
PCBs	50	0.005
Arsenic	18	0.002
Barium	251	0.026
Cadmium	10	0.001
Chromium	35	0.004
Lead	1 200	0.12
Zinc	1 130	0.12

---

[1] Franklin Associates (1985). All values are 90th percentile concentrations except for the value reported for trichlorotrifluoroethane, which is a 75th percentile concentration.

[2] Based on 103.7 t/a "oil and grease".

defined (e.g., "organic chemicals", "heavy metal hydroxides") have been placed into the appropriate generic category. Based on these assumptions, the inorganic wastes can be categorized as follows:

Inorganic Wastes To Be Sent to the Physical/Chemical Treatment Plant	Quantity (t/a)
Non-hazardous material	79 600.0
Hazardous metals	1 318.0
Hazardous organics	540.0
Cyanide	22.9
Fluoride	0.07
Nitrate	1.6

Inorganic Wastes To Be Sent Directly to the Solidification Plant	Quantity (t/a)
Non-hazardous material	28 384.0
Hazardous metals	4 143.0
Hazardous organics	237.0
Fluoride	0.014
Nitrate	4.9

Most of the inorganic material characterized as "non-hazardous" in the wastes received by the physical/chemical plant is water; the greatest quantity of "non-hazardous" inorganic material to be sent directly to solidification is silica and calcium carbonate. The estimation of the concentrations of surrogate chemicals selected to represent the "hazardous" portions of the inorganic wastes are described in the next section.

B.4.2 ESTIMATES OF SURROGATE CHEMICAL QUANTITIES

The total quantities of hazardous "metals" and "organics" in the inorganic wastes have been represented in the risk assessment by those chemicals for which adequate toxicological data are currently available. For the organic category, the total quantity of "hazardous" material has been distributed among the surrogate chemicals using the formula:

$$Qsi = \frac{(Qi)(Qh)}{Qt}$$

Where:

Qsi = Quantity of surrogate chemical, t/a

Qi = Projected quantity of chemical to be received at the OWMC facility (as estimated using the CANVIRO and Franklin data bases), t/a

Qh = Total quantity of "hazardous" organics in the inorganic wastes; 237 t/a for the wastes to be sent directly to the solidification plant, 540 t/a for the wastes to be sent to the physical/chemical treatment plant.

Qt = Total projected quantity of those chemicals selected as surrogates (estimated using the CANVIRO and Franklin data bases); 163 t/a for the wastes to be sent directly to the solidification plant, 105 t/a for the wastes to be sent to the physical/chemical treatment plant.

The distribution of the "hazardous" metals was conducted in a similar fashion. The quantities of surrogate chemicals developed for the purposes of the risk assessment using the approach outlined above are presented for the inorganic wastes in Tables B-29 through 32.

TABLE B-29

QUANTITIES OF ORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Chemical	Qi[1] (t/a)	Qsi[2] (t/a)
Toluene	66.30	342.65
Phenols	20.20	104.40
Styrene	7.90	40.83
Acrylonitrile	7.90	40.83
Petroleum distillates	0.83	4.29
Cresols	0.40	2.07
Ethylene glycol	0.36	1.87
1,1,1-Trichloroethane	0.17	0.88
Xylene	0.16	0.83
Tetrachloroethylene	0.08	0.41
Trichlorotrifluoroethane	0.06	0.31
Trichloroethylene	0.04	0.21
Naphthalene	0.04	0.21
Dichlorodifluoromethane	0.03	0.16
Benzene	0.01	0.05
Benzo(a)Anthracene	0.002	0.01
PCBs	0.002	0.01
Benzo(a)Pyrene	0.001	0.005
	104.5	540.0

---

[1] Projected quantity of chemical in the inorganic waste streams to be received at the physical/chemical treatment plant (based on the CANVIRO and Franklin data bases).

[2] Quantity of surrogate chemical.

TABLE B-30

QUANTITIES OF INORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES TO BE SENT TO THE PHYSICAL/CHEMICAL TREATMENT PLANT

Chemical	Qi[1] (t/a)	Qsi[2] (t/a)
Cyanide	22.94	22.94
Fluoride	0.07	0.07
Nitrate	1.55	1.55
Zinc	416.16	753.43
Chromium	106.86	193.46
Copper	84.92	153.74
Nickel	80.24	145.26
Silver	16.35	29.60
Cadmium	8.54	15.46
Lead	6.93	12.54
Arsenic	3.73	6.75
Antimony	3.72	6.73
Mercury	0.43	0.77
Barium	0.01	0.02
Total Metals	727.9	1 317.8

---

[1] Projected quantity of chemical in the inorganic waste streams to be received at the physical/chemical treatment plant (as based on the CANVIRO and Franklin data bases).

[2] Quantity of surrogate chemical.

TABLE B-31

QUANTITIES OF ORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Chemical	Qi[1] (t/a)	Qsi[2] (t/a)
Phenols	75.81	110.7
Formaldehyde	42.72	62.4
Toluene	39.96	58.3
Petroleum distillates	2.65	3.9
Xylene	0.51	0.75
1,1,1-Trichloroethane	0.36	0.53
Tetrachloroethylene	0.17	0.24
Trichlorotrifluoroethane	0.13	0.19
Cresols	0.10	0.15
Trichloroethylene	0.08	0.12
Naphthalene	0.08	0.12
Ethanol	0.07	0.10
Dichlorodifluoromethane	0.07	0.10
Benzene	0.03	0.04
PCBs	0.005	0.007
Benzo(a)Anthracene	0.004	0.006
Benzo(a)Pyrene	0.002	0.003
	162.8	237.0

---

[1] Projected quantity of chemical in the inorganic waste streams to be sent directly to the solidification plant (as based on the CANVIRO and Franklin data bases).

[2] Quantity of surrogate chemical.

TABLE B-32

QUANTITIES OF INORGANIC SURROGATE CHEMICALS IN THE INORGANIC WASTES TO BE SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Chemical	Qi[1] (t/a)	Qsi[2] (t/a)
Cyanide	1.03	1.03
Fluoride	0.01	0.01
Nitrate	4.91	4.91
Zinc	908.72	1 765.37
Lead	475.33	923.42
Copper	469.93	912.93
Chromium	156.70	304.43
Nickel	117.90	229.04
Cadmium	2.09	4.06
Mercury	1.36	2.63
Silver	0.51	0.98
Arsenic	0.04	0.08
Barium	0.03	0.05
TOTAL METALS	2 132.6	4 143.0

---

[1] Projected quantity of chemical in the inorganic waste streams to be sent directly to the solidification plant (as based on the CANVIRO and Franklin data bases).

[2] Quantity of surrogate chemical.

B.5 TRANSPORTATION SPILL SURROGATE SELECTION

B.5.1 INTRODUCTION

In order to assess the public health and environmental effects resulting from a transportation spill, surrogate chemicals were selected to represent the waste streams to be received by the proposed OWMC Facility. The starting point for the selection of surrogate chemicals was the CANVIRO data base, which had been developed to estimate the types and quantities of special wastes generated in Ontario (CANVIRO 1985). As previously described in Section B.1, this waste characterization data base has been categorized using the University of California at Davis (UCD) classification scheme.

The methodologies used for the selection of surrogate chemicals for spills onto the roadway and into surface water are discussed separately in the following sections.

B.5.2 TRANSPORTATION SPILL ONTO THE ROADWAY

The procedure used in selecting a surrogate chemical for the evaluation of a spill onto the roadway was previously presented in the Phase 4A Generic Risk Assessment (ENVIRON 1985). The selection procedure was based on a ranking scheme developed to identify the most hazardous chemical in any UCD category of waste to be received by the proposed OWMC Facility.

Each UCD category is comprised of many different waste streams which differ in terms of chemical constituents and concentrations. For purposes of the risk assessment, it was assumed that a truck load of special waste can be adequately represented by the average characteristics of a UCD category.

The ranking scheme developed in Phase 4A generated a hazard index to represent the degree of hazard associated with each chemical constituent of the waste streams of concern. The hazard index was based on three factors: (1) the toxicity of the constituents to be released to the environment; (2) the potential for the constituent to be released to the environment; and (3) the quantity of chemical available for release. The hazard index for each constituent can be calculated by adding together a set of scores for the following three factors:

$$HI = R + T + Q$$

Where:

HI = Hazard Index

R = Release Potential, as represented by a score for the chemical's vapor pressure

T = Toxicity, as represented by a score for the chemical's acute toxicity or LD_{50}*

Q = Quantity of chemical available for release, as represented by a score

---

* The LD_{50} represents the dose which produces fatality in 50 percent of the exposed population.

The hazard indices calculated for the various UCD categories of waste are shown on Table B-33. As shown, the highest hazard indices were calculated for UCD categories for which methylene chloride was selected as the surrogate chemical. Therefore, methylene chloride was chosen as the surrogate chemical for transportation spills onto land.

The final step characterizing the composition of the wastes spilled in the event of a spill onto the roadway was to calculate the mass fraction of the surrogate chemical, methylene chloride, in the UCD category. The mass fraction was previously defined in the Phase 4A Generic Risk Assessment (ENVIRON 1985), as simply the average concentration of a chemical in a UCD class. The mass fractions for methylene chloride in the two UCD categories of highest hazard are as follows:

UCD	Mass Fraction
211	0.62
213	0.68

In the risk assessment, the highest mass fraction (0.68) was used to estimate releases of methylene chloride resulting from a transportation spill onto the roadway.

TABLE B-33

HAZARD INDICES FOR SPILL ONTO THE ROADWAY

UCD Category	Constituent	Hazard Index
211	Methylene chloride	19
212	Trichloroethylene	16
213	Methylene chloride	19
214	Acetone	16
215*	Toluene	16
	Acetone	16
216*	Acetone	15
	Toluene	15
	Methyl Ethyl Ketone	15
217	Ethanol	16
224	Acrylonitrile	17
225	Formaldehyde	16
227*	Toluene	14
	Methyl Ethyl Ketone	14
241	Petroleum distillates	12
242	PCB	8
243	Ethanol	14
245	Formaldehyde	16
247	Methylene chloride	16
248	Ethanol	16
254	Methylene chloride	17
257	Formaldehyde	16
261	Styrene	15
262	Formaldehyde	15
263	Acrylonitrile	17
264	Petroleum distillates	12
281	Petroleum Distillates	12
283*	Trichloroethylene	14
	Benzene	14
286	Phenol	11
289	Petroleum distillates	12
416	Ethanol	14
446	Trichloroethylene	17
447	Tetraethyl lead	10
454*	Petroleum distillates	15
	Toluene	15
455*	Petroleum distillates	13
	Toluene	13
513	Acetone	15
521	Toluene	12
523	Formaldehyde	16
531*	Petroleum distillates	14
	Acetone	14

---

* The same hazard index was calculated for two chemicals in the UCD.

B.5.3 TRANSPORTATION SPILL INTO SURFACE WATER

In Phase 4B, the analysis of transportation spills has been extended to include the impacts from a transportation spill into surface water. The procedure used for selecting surrogate chemicals for spills into surface water in Phase 4B is somewhat different from that previously presented for selecting surrogate chemicals for spills onto land. As with the analysis of the spill onto land, the starting point for the evaluation of a spill into a stream was the waste characterization data developed by CANVIRO.

In the risk assessment, it was assumed that each waste stream would be transported separately and that a waste stream is best represented by the constituents in a UCD category. To select the surrogate chemicals for transportation releases to surface water, a ranking scheme was developed to identify the UCD category of waste that may be received by the proposed facility. The hazard ranking was based on two factors: (1) toxicity of the constituent, and (2) the quantity of chemical available for release. A hazard index for each individual constituent in the UCD categories was calculated as follows:

HI = Q/T

HI = Hazard Index

Q = Quantity, as represented by the mass fraction

T = Toxicity, as represented by the LD_{50}

The hazard index calculated for UCD 213 is presented in Table B-34 as an illustration of this approach.

The next step in the surrogate selection process was the calculation of an overall hazard index for each UCD category of concern. The overall hazard index was calculated by summing the hazard indices for the individual contaminants within the UCD. In this way the hazard indices for each UCD category were ranked in order to determine the UCD category of highest hazard, as shown on Table B-35.

The UCD class with the highest overall hazard index is UCD 447, tetraethyl lead sludge. However, this UCD category was eliminated from consideration in this analysis since the total quantity of the UCD category predicted to be received at the facility is only 5 t/a. Even assuming that this waste is all received in one shipment, the quantity is significantly less than the capacity of a tank truck (20 tonne). Therefore, the waste stream of highest hazard was determined to be UCD 213.

Chemicals that spill into water can be generally classified into three main categories: (1) "mixers", soluble compounds that mix with water very rapidly; (2) "floaters", immiscible (slightly soluble or insoluble) compounds that float on water; and (3) "sinkers", immiscible compounds that sink to the bottom of a water course (Thibodeaux 1980). To account for

TABLE B-34

HAZARD INDEX FOR UCD 213

Contaminant	Mass of Contaminant	Mass Fraction	LD_{50}*	Hazard Index
Methylene chloride	61.1	0.13844	167	8.29 E-04
1,1,1-Trichloroethane	56.9	0.12901	750	1.72 E-04
Petroleum distillates	29.0	0.06587	1 240	5.31 E-05
Phenols	4.1	0.00938	300	3.13 E-05
Chloroform	8.3	0.01882	800	2.35 E-05
Dichlorobenzene	4.7	0.01060	414	2.56 E-05
Trichloroethylene	20.8	0.04723	4 920	9.60 E-06
Methyl ethyl ketone	8.3	0.01869	2 740	6.82 E-06
Carbon tetrachloride	7.4	0.01673	2 800	5.97 E-06
Tetrachloroethylene	20.8	0.04723	8 100	5.83 E-06
Pyridine	2.1	0.00480	891	5.39 E-06
Ethanol	11.3	0.02561	5 560	4.61 E-06
Methyl isobutyl ketone	4.1	0.00932	2 080	4.48 E-06
Styrene	0.5	0.00123	316	3.91 E-06
Chlorobenzene	4.7	0.01060	2 830	3.75 E-06
Xylene	6.1	0.01375	4 300	3.02 E-06
Acetone	8.3	0.01869	9 750	1.92 E-06
Benzene	2.7	0.00603	3 800	1.59 E-06
Trichlorotrifluoroethane	22.9	0.05196	43 000	1.21 E-06
Toluene	2.7	0.00603	5 000	1.21 E-06
Isopropyl alcohol	2.1	0.00480	5 840	8.22 E-07
Naphthalene	0.5	0.00123	1 780	6.94 E-07
Ethylene glycol	2.1	0.00480	7 500	6.40 E-07
Hexachlorobenzene	2.6	0.00580	10 000	5.80 E-07
n-Hexane	2.5	0.00571	28 710	1.99 E-07
Urethane	0.2	0.00044	2 500	1.77 E-07
Chlorinated paraffins	0.9	0.00209	NA	NA
Oil and grease	31.4	0.07110	NA	NA
Lacquer	3.6	0.00815	NA	NA
Solids and water	107.3		NA	NA
	440.			1.20 E-03

---

* The LD_{50} represents the dose which produces fatality in 50 percent of the exposed population.

TABLE B-35

OVERALL HAZARD INDEX FOR INDIVIDUAL UCD CATEGORIES

UCD Category	Sum Hazard Index
447	1.8 E-03
213	1.2 E-03
211	7.7 E-04
242	6.1 E-04
212	6.0 E-04
215	5.4 E-04
513	4.7 E-04
264	4.5 E-04
523	4.2 E-04
217	3.3 E-04
254	3.3 E-04
216	3.3 E-04
214	3.0 E-04
248	2.9 E-04
243	1.7 E-04
245	1.5 E-04
241	1.3 E-04
263	1.2 E-04
261	1.1 E-04
454	1.0 E-04
531	1.0 E-04
446	9.5 E-05
227	8.7 E-05
257	7.4 E-05
521	6.3 E-05
224	6.1 E-05
247	5.9 E-05
512	5.7 E-05
281	4.6 E-05
262	3.6 E-05
225	2.7 E-05
416	2.2 E-05
455	2.9 E-06
252	1.7 E-06
289	1.3 E-06
282	2.6 E-07

the fact that chemicals in the wastes to be transported to the proposed OWMC facility may act differently when spilled in water, one chemical from each of the above categories was selected from UCD 113 for analysis.

The chemicals reported in UCD 213 were categorized according to whether they would be expected to float, sink, or mix when spilled into a stream, as shown in Table B-36. For each of these categories, the chemical of highest hazard was chosen for analysis:

Chemical Type	Surrogate
Mixer	Pyridine
Floater	Petroleum distillates
Sinker	Methylene chloride

In the current analysis, the entire quantity of waste spilled into the stream was conservatively assumed to consist of the surrogate chemical.

TABLE B-36

CLASSIFICATION OF CHEMICALS IN UCD 213

Chemical	Hazard Index	Behavior
Methylene chloride	8.3 E-04	Sinks
1,1,1-Trichloroethane	1.7 E-04	Sinks
Petroleum distillates	5.3 E-05	Floats
Phenols	3.1 E-05	May float or sink
Chloroform	2.4 E-05	Sinks
Dichlorobenzene	2.6 E-05	Sinks
Trichloroethylene	9.6 E-06	Sinks
Methyl ethyl ketone	6.8 E-06	Floats and mixes slowly
Carbon tetrachloride	6.0 E-06	Sinks
Tetrachloroethylene	5.8 E-06	Sinks
Pyridine	5.4 E-06	Mixes
Ethanol	4.6 E-06	Floats and mixes
Methyl isobutyl ketone	4.5 E-06	Floats and mixes slowly
Styrene	3.9 E-06	Floats
Chlorobenzene	3.8 E-06	Sinks
Xylene	3.2 E-06	Floats
Acetone	1.9 E-06	Floats and mixes
Benzene	1.6 E-06	Floats
Trichlorotrifluoroethane	1.2 E-06	Sinks
Toluene	1.2 E-06	Floats
Isopropyl alcohol	8.2 E-07	Floats and mixes
Naphthalene	6.9 E-07	Solidifies and floats or sinks
Ethylene glycol	6.4 E-07	Sinks and mixes
Hexachlorobenzene	5.8 E-07	Floats or sinks
n-Hexane	2.0 E-07	Floats
Urethane	1.8 E-07	Unknown

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APPENDIX C

CHARACTERIZATION OF CONTAMINANT RELEASES

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APPENDIX C

CHARACTERIZATION OF CONTAMINANT RELEASES

C.1 INTRODUCTION

The proposed OWMC special waste management facility will consist of four major components:

- An incinerator for the combustion of organic wastes;
- A physical/chemical treatment plant for the processing of inorganic and aqueous organic wastes;
- A solidification plant for stabilizing inorganic wastes received from off-site generators and solid residues from the incinerator and the physical/chemical treatment plant; and
- An engineered landfill for the disposal of stabilized solid wastes from the solidification plant.

Associated waste reception, handling, and storage facilities will also be provided (see Monenco 1987).

Releases of potentially hazardous contaminants to the off-site environment are expected to occur during normal operation of the OWMC facility. The specific routine releases evaluated in the current risk assessment are the following:

- Incinerator stack emissions;
- Evaporator stack emissions;
- Leachate from the engineered landfill; and
- Fugitive emissions associated with
  - bulk waste unloading and transfer,
  - routine leaks and spills,
  - drum handling,
  - waste storage, and
  - physical/chemical treatment.

Contaminants may also be released to the environment as a result of facility upsets, malfunctions, and accidents, or in the event of an off-site transportation accident. Emissions resulting from the following upsets, malfunctions, and accidents have been evaluated in the risk assessment for the OWMC facility:

- A tank farm fire;
- Mixing of incompatible wastes;
- Incinerator scrubber failure;
- pH excursion during the treatment of cyanide-containing wastes; and
- pH excursion during sulfide precipitation of heavy metals
- A transportation spill resulting in the release of wastes
  - onto the roadway, or
  - into surface water.

The nature and magnitude of potentially hazardous releases resulting from the operation of the OWMC facility have been estimated in the risk assessment using a combination of empirical data generated at comparable facilities (see ORF 1987; Abkowitz, Eiger, and Srinivasan 1984) and engineering calculations (see Monenco 1987; M.M. Dillon 1984; M.M. Dillon 1985). The calculations used to estimate contaminant emission rates for the purposes of the risk assessment are presented in this appendix.

C.2 ROUTINE EMISSIONS TO THE ATMOSPHERE

C.2.1 INCINERATOR STACK

A rotary kiln, high temperature incinerator will be used at the OWMC facility to treat potentially hazardous organic wastes. Flue gases generated during the combustion process will be treated by a system consisting of a spray dryer, an electrostatic precipitator, an induced draft fan and a three stage wet gas scrubber. Routine incinerator stack emissions may include the following types of toxic constituents:

- Uncombusted residues of organic chemicals in feed;
- Metals present in the waste feed;
- Acid gases generated by combustion; and
- Products of incomplete combustion.

In the risk assessment, all incinerator stack emission rates have been based on the following assumptions (Monenco 1987):

- A facility capacity of 150 000 t/a, corresponding to the operation of a single incinerator unit with a total design throughput of 4.76 t/hr;
- The incinerator will be available for operation 7 000 hours out of 8 760 hours per year;
- A total average flue gas rate of 22 860 Nm^3/hr (dry, 7% O_2).

Therefore, all emission projections provided on a flue gas concentration basis (mass/volume) have been converted to a rate basis (mass/time) using the following formula:

$$Q_i = \frac{(C_i)(F)(t_o)}{3600\ (t_T)}$$

Where:

Q_i = emission rate for chemical i, g/s

C_i = projected concentration in the flue gas, g/Nm^3

F = flue gas emission rate, 22 860 Nm^3/hr

t_o = period of operation, 7 000 hr/a

t_T = hours in a year, 8 760 hr/a

The approaches taken for the estimation of emission rates for the residues of organic waste constituents, metals, acid gases, and products of incomplete combustion are described separately in the following sections of this appendix.

C.2.1.1 Uncombusted Organic Residues

Residues of the organic feed to the OWMC incinerator are expected to be present in the stack emissions since the destruction of the wastes by the combustion process will not be 100 percent effective. Destruction and removal efficiencies (DREs) of 99.99 percent or better are typically achieved by similar facilities (Midwest Research Institute 1984). To evaluate the potential health risks associated with the release of organic feed residues from the incinerator stack, the nature and magnitude of these emissions were estimated.

The total emission rates for organic feed residues were based on an expected stack gas organic carbon concentration of 15 mg/Nm^3 (see Monenco 1987, 1986d). The total organic emission rate is related to the organic carbon emission rate for a given chemical as follows:

$$TH = \frac{OC \times MW}{CW}$$

Where:

TH = Total organic emission rate, mg/Nm^3

OC = Organic carbon emission rate, mg/Nm^3

MW = Molecular weight, g/mol

CW = Carbon weight (weight of carbon in the constituent), g/mol

For example, if trichloroethylene were the only organic contaminant in the stack effluent, and an organic carbon emission rate of 15 mg/Nm^3 was measured, the total organic emission rate could be calculated as follows:

$$TH = \frac{15 \text{ mg/Nm}^3 \times 131.4 \text{ g/mol}}{24.0 \text{ g/mol}}$$

$$TH = 82.1 \text{ mg/Nm}^3$$

The constituents of the organic waste were examined to estimate the molecular weight and carbon weight of the organic residues in the stack effluent. Accounting for the presence of products of incomplete combustion (PIC) in the stack gases (estimated as approximately 0.5 mg/Nm^3 organic carbon - see Section C.2.1.4), the total organic emission rate associated with uncombusted residues of organic feed to the incinerator was calculated as follows:

$$TH = (OC-OCp) \div \textstyle\sum_i [(F_i)(CW_i/MW_i)]$$

$$TH = (15 - 0.5) \div (0.53)$$

$$TH = 27.5 \text{ mg/Nm}^3$$

Where:

TH = Total organic emissions due to uncombusted feed (excluding PICs), mg/Nm^3

OC = Expected organic carbon emissions, 15 mg/Nm^3

OC_p = Expected organic carbon emission for PICs, 0.5 mg/Nm^3

F_i = Weight fraction in feed, constituent i, g/g (see Appendix B)

MW_i = Molecular weight, constituent i g/mol

CW_i = Carbon Weight, constituent i, g/mol

The identity and quantity of specific organic waste constituents in the stack emissions was based on information derived primarily from the CANVIRO data base (CANVIRO 1985). This data base was used in the risk assessment to select "surrogate" chemicals to represent the total quantity of potential hazardous material to be fed to the incinerator. (See Appendix B for a discussion of the surrogate selection process.) The following formula was used to predict incinerator stack emission rates for each surrogate chemical:

$$C_i = TH \times \frac{Qs_i}{Qt}$$

Where:

C_i = Estimated concentration of chemical i in the incinerator stack emissions, mg/Nm^3

TH = Total organic emission rate, 27.5 mg/Nm^3

Qs_i = Quantity of surrogate chemical i in the incinerator feed, t/a

Qt = Total quantity of all surrogates in the incinerator feed, t/a

The emission concentration calculated for polychlorinated biphenyls (PCBs) using the above equation was divided by 100 to account for the higher DRE that will be required for this chemical. (A DRE of 99.9999 percent is typically required for PCBs, which is 100 times greater than the DRE of 99.99 percent for other hazardous constituents in the feed of hazardous waste incinerators. OWMC is committed to achieving this higher level of destruction and removal for PCBs.) Emission rates for the surrogate chemicals were calculated by multiplying the estimated concentration in the stack effluent by the total volumetric flue gas rate, 22860 Nm^3/hr (dry, 7 percent O_2), using the formula introduced in Section C.2.1. The predicted emission rates for the surrogate chemicals are summarized in Table C-1.

It should be noted that the approach outlined above results in emission rates corresponding to destruction and removal efficiencies (DREs) of approximately 99.9 percent for all compounds other than PCBs, where a DRE of 99.999 percent was estimated. Therefore, the predicted emission rates for organic constituents of the feed are somewhat higher than would be calculated assuming DREs that have been achieved at existing hazardous waste incinerators, and higher than would be expected at the OWMC facility.

C.2.1.2 Metals

The special waste to be treated in the OWMC incinerator will be primarily organic in nature. However, relatively small quantities of inorganic substances, such as metals, are also expected to be constituents of these wastes. Although the largest portion of most of these metals would be released from the incinerator in solid form along with slag and ash, a fraction is expected to be vented to the atmosphere from the stack.

TABLE C-1

PROJECTED INCINERATOR EMISSIONS - ORGANIC FEED RESIDUES

Chemical Constituent	Organic Incinerator Emission (g/sec)
Acetone	1.24 E-02
Acrylonitrile	7.71 E-05
Benzene	8.67 E-04
Benzo(a)anthracene	5.83 E-06
Benzo(a)pyrene	2.33 E-06
Butadiene, 1,3-	6.87 E-06
Carbon tetrachloride	3.90 E-04
Chloroform	6.55 E-04
Ethanol	1.82 E-02
Ethylene glycol	2.29 E-02
Ethylene oxide	5.87 E-06
Formaldehyde	2.08 E-04
Hexachlorobenzene	3.31 E-06
Hydrazine	1.92 E-08
Methyl ethyl ketone	3.34 E-03
Methylene chloride	6.43 E-03
PCB	8.31 E-06
Petroleum distillates	2.00 E-02
Pyridine	3.86 E-04
Tetrachloroethylene	9.71 E-03
Tetraethyl lead	1.54 E-06
Toluene	1.73 E-02
Trichloroethane,1,1,1-	6.75 E-03
Trichloroethylene	8.63 E-03
Xylene	8.79 E-03

The Ontario Research Foundation (ORF) has conducted a review of metal emission data from existing hazardous waste incineration facilities. (ORF 1987). The data compiled by ORF are summarized in Table C-2. For the purposes of the risk assessment, the highest emission rate observed for each metal has been conservatively assumed in predicting releases from the proposed OWMC incinerator. Where information on the gas conditions corresponding to the reported metal concentration was available, conversion to dry, 7 percent O_2 conditions were made when appropriate.

The oxidation state of chromium, a substance to be emitted from the OWMC incinerator, is a critical issue in characterizing metal emissions for the risk assessment. Chromium compounds can exist in any of three oxidation states: elemental (Cr), trivalent (Cr III), or hexavalent (Cr VI). The toxicity of chromium varies considerably from one form to another. Cr VI is the most hazardous form of chromium; it is generally considered to be one of the most potent human carcinogens via inhalation exposure. In contrast, Cr and Cr III have not been shown to be carcinogenic in either humans or laboratory animals.

Studies performed at two municipal solid waste (MSW) incineration facilities indicate that less than 1 percent of the total chromium emitted from these sources is in the

TABLE C-2

PROJECTED INCINERATOR EMISSIONS - METALS

Metal Constituent	Observed Emission Concentrations[1] ($\mu g/m^3$)	Projected Emission Rates[2] (g/sec)
Arsenic	1.0-5.9	3.00 E-05
Beryllium	0.007-0.04	2.04 E-07
Cadmium	0.05-60	4.51 E-04
Chromium	1.6-140	1.07 E-03
Copper	130-500	2.52 E-03
Lead	15-3 000	1.52 E-02
Mercury	0.1-100	5.19 E-04
Nickel	1.6-60	3.04 E-04
Zinc	4.3-7 000	3.56 E-02

---

[1] Source: ORF 1987. Gas conditions unknown for all metals except cadmium and chromium, which are reported at 11.6 percent O_2. Emission rates for these two metals were therefore multiplied by 1.49, [(21-7)/(21-11.6)], to convert to 7 percent O_2.

[2] Derived using the formula introduced in Section C.2.1 and the highest observed emission concentration.

hexavalent state (EPA, 1986a; EPA, 1986b). Furthermore, hexavalent chromium emitted from a specialty steel plant, under conditions likely to enhance the release of Cr VI, has been reported at a level equivalent to approximately 4% of the total chromium (EPA 1985). Although the installations tested to date are quite different than the proposed OWMC facility in both design and operation, the analysis results do indicate that hexavalent chromium generally comprises less than 10 percent of the chromium emitted from combustion sources.

Theoretical arguments also tend to suggest that the greatest quantity of chromium emitted will be in the trivalent state. Environmental Elements Corporation and Von Roll, the incinerator technology consultants to OWMC, have estimated that approximately 1 percent of the total chromium emissions from the facility will be in the hexavalent form. This estimate is based, in part, on the fact that hexavalent chromium is a strong oxidizing agent which is readily converted to the trivalent form in the presence of reducing agents. In the acid gas scrubbing system proposed for the OWMC facility, hexavalent chromium will be exposed to reducing compounds (such as sulfur dioxide) that are dissolved in the scrubber water, and therefore be transformed to Cr III.

Based on available information, it is unlikely that Cr VI will comprise greater than 10 percent of the total chromium emissions. It has therefore been assumed in the risk assessment that 10 percent of the chromium will be emitted as Cr VI, and 90 percent as Cr III.

C.2.1.3 Acid Gases

A variety of acid gases, including hydrogen chloride (HCl), hydrogen fluoride (HF), sulfur dioxide (SO_2), and nitrogen oxides (NO_x), may be formed during the incineration of hazardous wastes. The extent to which these acid gases will be generated and released from an incineration facility is dependent on the waste composition, incinerator design and operation, and the effectiveness of the proposed pollution control equipment. EEC/Von Roll, designers of the proposed OWMC incinerator, have provided expected emission rates for HCl, HF, SO_2, and NO_x (Monenco 1987, 1986d). These projections were used in the Phase 4B risk assessment and are presented in Table C-3.

C.2.1.4 Products of Incomplete Combustion

Products of Incomplete Combustion (PICs), such as polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF), may be formed during the incineration of hazardous waste. These compounds, which by definition are not present in the waste feed, must be considered in evaluating the potential health risks associated with incinerator stack emissions.

TABLE C-3

PROJECTED INCINERATOR EMISSIONS - ACID GASES

Constituent	Expected Emission Concentrations[1] (mg/m^3)	Projected Emission Rates[2] (g/sec)
Hydrogen Chloride	15	7.63 E-02
Hydrogen Fluoride	1	5.19 E-03
Nitrogen Oxides	200	1.01
Sulfur Dioxide	150	7.63 E-01

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[1] Source: Monenco 1987, ORF 1987.

[2] Derived using the formula introduced in Section C.2.1.

Data on the long-term emission of PCDD and PCDF from hazardous waste incinerators similar in design and operation to that proposed by OWMC have been compiled and reviewed by the Ontario Research Foundation (ORF 1987). Of the facilities included in the review, HIM Biebesheim in West Germany and SAKAB in Sweden are most similar in design and operation to the incinerator proposed by OWMC. Therefore, data from these two facilities were considered by ORF to best reflect potential PCDD/PCDF emissions from the OWMC incinerator. Based on their review, ORF has recommended the use of one particular set of PCDD/PCDF emission data collected at the HIM facility in the risk assessment (ORF 1987). These data, and the projected PCDD/PCDF emissions from the proposed OWMC incinerator, are presented in Table C-4.

PCDD/PCDF emission rates are often expressed in terms of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) equivalents, or TEQ. By using the TEQ approach, the emission rates for the various forms of PCDD/PCDF are represented by a single species, 2,3,7,8-TCDD. Many different methodologies for converting PCDD/PCDF emission rates to TEQ emission rates have been proposed. In the risk assessment, TEQ emissions have been calculated using methodologies developed by the Ontarion Ministry of the Environment (OMOE) and the United States Environmental Protection (USEPA). Based on the OMOE approach, a TEQ emission rate of 6.4×10^{-8} g/s has been estimated; a

TABLE C-4

PROJECTED INCINERATOR EMISSIONS - POLYCHLORINATED DIBENZO-p-DIOXINS (PCDD) AND POLYCHLORINATED DIBENZOFURANS (PCDF)

Constituent	Observed Emission Concentrations[1] (ng/m^3)	Projected Emission Rates[2] (g/sec)
PCDD		
Tetra-		
2378-	0.04	2.3 E-10
non 2378-	0.80	4.1 E-09
Penta-		
2378-	0.12	6.0 E-10
non 2378-	1.07	5.4 E-09
Hexa-	2.24	1.1 E-08
Hepta-	2.53	1.3 E-08
Octa-	7.30	3.7 E-08
Subtotal	14.11	7.2 E-08
PCDF		
Tetra-		
2378-	0.55	2.8 E-09
non 2378-	6.45	3.3 E-08
Penta-		
2378-	1.25	6.4 E-09
non 2378-	12.01	6.1 E-08
Hexa-	19.52	9.9 E-08
Hepta-	14.45	7.3 E-08
Octa-	8.79	4.5 E-08
Subtotal	63.03	3.2 E-07
TOTAL		3.9E-07

---

[1] Emission data from NUKEM HIM Report - June, 1985. Recommended for Phase 4B Risk Assessment (ORF 1987). Gas conditions reported as dry, 11.6% oxygen and converted to dry, 7% oxygen.

[2] Based on 22860 Nm^3/hr (dry, 7% oxygen) 150 000 t/a facility, operational 7 000 hr/a.

TEQ emission rate of 3.1 x 10^{-9} g/s has been calculated using the USEPA methodology. A detailed discussion of the concept of TEQ emission rates, and the calculations used in estimating TEQ emissions for the risk assessment, are included in Appendix H.

Both vapor-phase and particulate-bound PCDD/PCDF emissions have been detected at existing incineration facilities (see, for example, Cavallaro et al. 1982, Brocco et al. 1984, Ballschmiter et al. 1985, and Environment Canada 1986). The distribution of PCDD/PCDF between vapor-phase and particulate-bound PCDD/PCDF has varied greatly in the various investigations conducted. For example, while the results reported by Environment Canada (1986) show that virtually all PCDD/PCDF emitted from the Quebec municipal waste incinerator are adsorbed to particulate, the data gathered by Brocco et al. indicate that PCDD/PCDF may be emitted primarily in the vapor phase.

It should be recognized, however, that available flue gas sampling methodologies may not be adequate for the purposes of distinguishing between vapor-phase and particulate-bound PCDD/PCDF in the flue gas. Adsorption and desorption that occurs in the sampling equipment may lead to reported PCDD/PCDF distributions very different from those that actually exist in the flue gas. For this reason, the distribution between vapor-phase and particulate-bound PCDD/PCDF emitted from the

OWMC incinerator has been estimated on a theoretical basis. According to the designers of the proposed OWMC incinerator, the following temperature profile will exist in the post-combustion zone (Monenco 1986d):

- Boiler outlet: 325°C
- Spray drier outlet: 160°C
- Electrostatic precipitator: 160°C
- Quench: 63°C
- Stack: 130°C

Based on the vapor pressure of PCDD/PCDF, Teller and Lauber (1983) have developed the following table summarizing the effect of temperature on the vapor phase concentration of PCDD/PCDF:

Temperature (°C)	Percent Reduction Compared to 220°C
220°C	0
120°C	97-98
110°C	98-99
100°C	99-99.5

Therefore, a temperature reduction to approximately 100°C within the emission control system will result in the condensation of approximately 99 percent of the vapor phase PCDD/PCDF. It can therefore be assumed that vapor phase PCDD/PCDF emissions from the quench section of the pollution control equipment will be negligible. Furthermore, according to investigations conducted by Shaub (1985), desorption of

PCDD/PCDF from particulate at the higher temperatures in the stack should not be significant. Therefore, it is likely that the PCDD/PCDF released from the OWMC incinerator will be predominantly adsorbed to particulate.

Only limited testing for PICs other than PCDD/PCDF has been conducted at existing hazardous waste incineration facilities. The most complete data available were collected at various types of hazardous waste incinerators in the United States, and have been published by the Midwest Research Institute (MRI 1984).

Ideally, data generated at rotary kiln units similar to OWMC in design and operation would be used in projecting PIC emissions. However, since PIC emissions data specific to rotary kiln units are largely unavailable, the data compiled by MRI for a broader range of incinerator types were used to predict emission rates in the risk assessment. These data, while limited, are nonetheless the best available at the present time. Lacking data on the average concentrations of the various PICs in the stack gases, projected emission rates have been based on the midpoint of the values reported by MRI. Furthermore, only those PICs detected by MRI at more than one of the eight facilities tested are considered in this report. The PIC emission data from hazardous waste incinerators reported by MRI are summarized in Table C-5 along with projections developed for use in the Phase 4B risk assessment.

Based on the midpoint of the emission concentration values in Table C-5, the total emission concentration for the products of incomplete combustion has been projected as approximately 1.2 mg/Nm^3. This concentration can be related to the organic carbon emission rate associated with the PICs as follows:

$$OCp = THp \times \sum [(E_i/THp)(CW_i/MW_i)]$$

$$OCp = 1.2 \text{ mg/Nm}^3 \times 0.4$$

$$OCp = 0.5 \text{ mg/Nm}^3$$

Where:

OCp = Organic carbon emissions rate, PICs, mg/Nm^3

THp = Total organic emission rate, PICs, mg/Nm^3

E_i = Emission rate, constituent i, mg/Nm^3

CW_i = Carbon weight, constituent i, g/mol

MW_i = Molecular weight, constituent i, g/mol

This value of the organic carbon associated with PIC emissions has been used previously in calculating the projected quantity of feed residues in the stack emissions (see Section C.2.1.1).

C.2.2 EVAPORATOR UNIT

The physical/chemical plant will consist of two separate processing systems for the treatment of inorganic and aqueous organic wastes. Chemicals present in these wastes will be:
(a) destroyed or stabilized within the physical/chemical plant;
(b) separated and solidified for disposal in the engineered

TABLE C-5

PROJECTED INCINERATOR EMISSIONS - PICs OTHER THAN PCDD/PCDF

Chemical Constituent	Observed Emission Concentrations[1] (μg/m^3)	Projected Emission Rates[2] (g/sec)
Benzene	12-670	1.7 E-03
Bromodichloromethane	3-32	8.9 E-05
Bromoform	0.2-2.4	6.7 E-06
Chlorobenzene	1-10	2.8 E-05
Chloroform	1-1 330	3.4 E-03
Methyl Ethyl Ketone	3	1.5 E-05
Methylene Chloride	2-27	7.3 E-05
Naphthlene	5-100	2.7 E-04
Nitrophenol,o-	25-50	1.9 E-04
Phenol	4-22	6.6 E-05
Tetrachloroethylene	0.1-2.5	6.6 E-06
Toluene	2-75	2.0 E-04
Trichloroethane, 1,1,1-	0.1-1.5	4.1 E-06

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[1] Source: MRI 1984.

[2] Derived using the formula introduced in Section C.2.1.

landfill; (c) separated for combustion in the rotary kiln incinerator; or (d) discharged in the liquid effluent sent to an evaporation unit. A portion of the vapors generated by the evaporation unit will be emitted to the atmosphere through a 45 meter stack. Although these emissions will consist primarily of water vapor, contaminants present in the liquid discharged to the evaporator may also be released.

The prediction of contaminant emission rates for the evaporator involved the following two steps:

- Analysis of the performance of the various physical/chemical processes in treating specific chemicals; and
- Calculation of the emission rates for chemicals which are ultimately discharged to the evaporation unit.

The approaches used in these steps are described in this section.

C.2.2.1 Physical/Chemical Treatment Plant Performance

As previously noted, inorganic and aqueous organic wastes will be processed separately in the physical/chemical treatment plant. Some or all of the following unit operations will be used to treat aqueous organic wastes:

- Bulk separation;
- Emulsion breaking/stripping;
- Dissolved air flotation; and
- Polishing with activated carbon filtration/reverse osmosis.

Inorganic wastes will be treated by some or all of the following processes:

- Bulk separation;
- Aqueous organic treatment processes, such as stripping;
- Oxidation;
- Coagulation/precipitation; and
- Polishing with activated carbon filtration and reverse osmosis.

The treatment performance of a given unit operation is dependent upon the chemical concentrations in the waste stream under consideration. For example, if a chemical is present at a concentration greater than its solubility limit in water, it can be readily removed through bulk separation. Since chemical concentration is an important factor in predicting the performance of the various processing units, the physical/chemical treatment operations were evaluated by separately analyzing those streams reported in the CANVIRO data base to contain specific organic contaminants (see Appendix B for a discussion of the CANVIRO data base).

To evaluate the performance of the physical/chemical treatment plant, a group of surrogate chemicals was selected to represent the hazardous organic portion of the inorganic and aqueous organic wastes. The methodology used to select the surrogate chemicals is described in Appendix B. The quantity of each stream containing one of the surrogate chemicals was prorated to account for the amount of potentially hazardous organic material in the wastes received at the physical/ chemical treatment plant. As shown in Appendix B, the appropriate prorating factors were calculated as follows:

$$f = \frac{Q_{hl}}{Q_t}$$

Where:

f = prorating factor

Q_{hl} = quantity of potentially hazardous material in the wastes, t/a

Q_t = total quantity of those chemicals selected as surrogates (estimated using the CANVIRO and Franklin data bases), t/a

For the aqueous organic streams, Q_{hl} = 155 t/a and Q_t = 80 t/a; and therefore f_a = 1.9. For the inorganic streams, Q_{hl} = 540 t/a, Q_t = 104.5 t/a; and therefore f_I = 5.2.

Using these factors, the prorated quantities of those inorganic and aqueous organic waste streams containing specific organic constituents were calculated. These quantities are shown in Tables C-6 and C-7.

The concentrations of specific chemicals in the inorganic and aqueous organic trains following each unit operation were estimated by the engineering consultants to OWMC (Monenco 1987a). These estimates, which are presented in Tables C-8 and C-9, were based on a review of the scientific literature (see

TABLE C-6

CONTAMINANT CONCENTRATIONS IN SPECIFIC AQUEOUS ORGANIC STREAMS TO BE RECEIVED BY THE PHYSICAL/CHEMICAL TREATMENT PLANT*

Stream	Contaminant	Concentration (mg/l)	Prorated Stream Quantity (t/a)
A-1	Phenol	20 000	198.0
A-2	Phenol	20 000	12.7
A-3	Phenol	20 000	1478.2
A-4	Phenol	2 000	1868.7
A-5	Formaldehyde	20 000	39.9
A-6	Acrylonitrile	5 000	416.6
A-7	Butadiene	500	416.6
A-8	Styrene	5 000	416.6
A-9	Xylene	10 000	91.3
A-10	Xylene	3 000	1.7
A-11	Toluene	200 000	39.9
A-12	Toluene	320 000	0.7
A-13	Toluene	5 000	1.7
A-14	Toluene	500 000	186.9
A-15	Trichloroethylene	1 600	39.9
A-16	Trichloroethylene	2 600	0.7
A-17	Trichloroethylene	40	1.7
A-18	Tetrachloroethylene	1 600	39.9
A-19	Tetrachloroethylene	2 600	0.7
A-20	Tetrochloroethylene	40	1.7

---

* "Petroleum distillates" emissions could not be assessed since the contaminant group was not sufficiently well-defined for the purposes of treatability performance evaluation. It was determined, however, that even if the total quantity of "petroleum distillates" projected to be received at the physical/chemical treatment plant were to be released from the evaporator stack, such emissions would not pose a significant risk to public health.

Derived from CANVIRO 1985

TABLE C-7

CONTAMINANT CONCENTRATIONS IN SPECIFIC INORGANIC STREAMS TO BE RECEIVED BY THE PHYSICAL/CHEMICAL TREATMENT PLANT*

Stream	Contaminant	Concentration (mg/l)	Prorated Stream Quantity (t/a)
I-1	Phenol	1 000	7371.0
I-2	Toluene	10 000	5353.1
I-3	Phenol	5 000	5353.1
I-4	Styrene	5 000	8233.9
I-5	Acrylonitrile	5 000	8233.9
I-6	Phenol	1 000	137.4
I-7	Toluene	40 000	5024.9
I-8	Phenol	5 000	5024.9
I-9	Toluene	10 000	1741.7
I-10	Phenol	5 000	1741.7
I-11	Toluene	10 000	5226.8
I-12	Phenol	5 000	5226.8
I-13	Toluene	10 000	1741.7
I-14	Phenol	5 000	1741.7
I-15	Toluene	10 000	260.1
I-16	Phenol	10 000	260.1

---

* "Petroleum distillates" emissions could not be assessed since the contaminant group was not sufficiently well-defined for the purposes of treatability performance evaluation. It was determined, however, that even if the total quantity of "petroleum distillates" projected to be received at the physical/chemical treatment plant were to be released from the evaporator stack, such emissions would not pose a significant risk to public health. The emissions of two other surrogates, ethylene glycol and cresol, were also judged not to pose a risk based on the same rationale.

Derived from CANVIRO 1985

TABLE C-8

CONCENTRATIONS OF SPECIFIC CHEMICALS FOLLOWING EACH UNIT OPERATION OF THE AQUEOUS ORGANIC WASTE TREATMENT PROCESS

Stream	Contaminant	Concentration (mg/l) Bulk Removal	Emulsion Breaking, DAF	Activated Carbon/ Reverse Osmosis
A-1	Phenol	20 000	20 000	0.02
A-2	Phenol	20 000	20 000	0.02
A-3	Phenol	20 000	20 000	0.02
A-4	Phenol	2 000	2 000	0.02
A-5	Formaldehyde	20 000	0.5	0.5
A-6	Acrylonitrile	5 000	0.5	0.5
A-7	Butadiene	500	0.5	0.5
A-8	Styrene	300	10	0.1
A-9	Xylene	175	5	0.05
A-10	Xylene	175	5	0.05
A-11	Toluene	535	5	0.2
A-12	Toluene	535	5	0.2
A-13	Toluene	535	5	0.2
A-14	Toluene	535	5	0.2
A-15	Trichloroethylene	1 100	20	0.025
A-16	Trichloroethylene	1 100	20	0.025
A-17	Trichloroethylene	40	20	0.025
A-18	Tetrachloroethylene	150	20	0.05
A-19	Tetrachloroethylene	150	20	0.05
A-20	Tetrochloroethylene	40	20	0.05

Source: Monenco 1987a

TABLE C-9

CONCENTRATIONS OF SPECIFIC CHEMICALS FOLLOWING EACH UNIT OPERATION OF THE INORGANIC WASTE TREATMENT PROCESS

		Concentration (mg/l)				
Stream	Contaminant	Bulk Removal	Stripping	Oxidation	Coagulation/ Precipitation	Activated Carbon/ Reverse Osmosis
I-1	Phenol	1 000	1 000	10	5	0.02
I-2	Toluene	535	5	5	5	0.2
I-3	Phenol	5 000	5 000	10	5	0.02
I-4	Styrene	300	10	10	2	0.1
I-5	Acrylonitrile	5 000	0.5	0.5	0.5	0.5
I-6	Phenol	1 000	1 000	10	5	0.02
I-7	Toluene	535	5	5	2	0.2
I-8	Phenol	5 000	5 000	10	5	0.02
I-9	Toluene	535	5	5	2	0.2
I-10	Phenol	5 000	5 000	10	5	0.02
I-11	Toluene	535	5	5	2	0.2
I-12	Phenol	5 000	5 000	10	5	0.02
I-13	Toluene	535	5	5	2	0.2
I-14	Phenol	5 000	5 000	10	5	0.02
I-15	Toluene	535	5	5	2	0.2
I-16	Phenol	10 000	10 000	10	5	0.02

Source: Monenco 1987a

Monenco 1986c, 1987a) and studies commissioned by OWMC (Zenon 1987). As a result of bulk removal, pressure filtration, and solids removal, the flow of individual streams containing organics would be reduced, as shown in Tables C-10 and C-11.

The only organic chemicals that were not examined on an individual stream basis were those identified in the Franklin data base as possible constituents of waste oil. (See Appendix B for a discussion of the Franklin data base.) Since "oil and grease" is expected in a large percentage of the inorganic and aqueous organic wastes, it was not feasible to conduct a stream-by-stream evaluation. Therefore, the effectiveness of the physical/chemical treatment plant in removing specific contaminants in oil and grease, as estimated by the engineering consultants to OWMC (Monenco 1987a), was used to estimate chemical concentrations in the wastes to be sent to the evaporation unit. These concentrations are presented in Table C-12.

As previously noted, small quantities of metals originally present in the inorganic wastes may be entrained in the evaporator emissions. To evaluate the significance of such releases, the performance of the physical chemical treatment plant in removing carcinogenic metals (nickel, arsenic, cadmium, and chromium) from the effluent to the evaporator was examined.

TABLE C-10

FLOWS OF INDIVIDUAL AQUEOUS ORGANIC WASTE STREAMS

Stream	Prorated Quantity to the Physical/Chemical Plant (t/a)	Prorated Quantity to the Evaporation Unit (t/a)
A-1	198.0	198.0
A-2	12.7	12.7
A-3	1 478.2	1 478.2
A-4	1 868.7	1 868.7
A-5	39.9	39.9
A-6	416.6	416.6
A-7	416.6	416.6
A-8	416.6	414.6
A-9	91.3	90.4
A-10	1.7	1.7
A-11	39.9	31.9
A-12	0.7	0.5
A-13	1.7	1.7
A-14	186.9	93.5
A-15	39.9	39.8
A-16	0.7	0.7
A-17	1.7	1.7
A-18	39.9	39.8
A-19	0.7	0.7
A-20	1.7	1.7

TABLE C-11

FLOWS OF INDIVIDUAL INORGANIC WASTE STREAMS

Stream	Prorated Quantity to the Physical/Chemical Plant (t/a)	Prorated Quantity to the Evaporation Unit (t/a)
I-1	7 125.3	7 125.3
I-2	5 125.7	5 125.7
I-3	5 174.6	5 174.6
I-4	7 922.0	7 922.0
I-5	8 233.9	7 959.4
I-6	137.4	132.8
I-7	5 024.9	4 665.7
I-8	5 024.9	4 857.4
I-9	1 741.7	1 667.7
I-10	1 741.7	1 683.7
I-11	5 226.8	5 004.7
I-12	5 226.8	5 052.6
I-13	1 741.7	1 667.7
I-14	1 741.7	1 683.7
I-15	260.1	249.0
I-16	260.1	251.4

TABLE C-12

CONSTITUENTS OF "OIL AND GREASE" IN THE EFFLUENT TO THE EVAPORATION UNIT

Contaminant	Solubility in Water (mg/L)	Reference	Estimated Concentration after Polishing[5] (mg/L)
Benzo(a)anthracene	5.7×10^{-3}	(1)	1.4×10^{-5}
Benzo(a)pyrene	3.8×10^{-3}	(1)	4.0×10^{-5}
Benzene	$1.8 \times 10^{+3}$	(1)	0.015
Dichlorodifluoromethane	280	(2)	1.0
Naphthalene	30	(3)	1.0
PCBs	3.1×10^{-2}	(1)	1.5×10^{-5}
Tetrachloroethylene	150	(2)	0.05
Toluene	515	(2)	0.2
1,1,1-Trichloroethane	4.4×10^{-3}	(2)	1.0
Trichloroethylene	1.1×10^{-3}	(2)	0.025
Trichlorotrifluoroethane	280	(4)	1.0
Xylene	187	(2)	0.05

Sources: (1) Mabey 1982
(2) Verscheuren 1983
(3) Perry and Chilton 1970
(4) General Electric
(5) Monenco 1987b.

According to estimates provided by the engineering consultants (Monenco 1987a), most metals would be removed during the coagulation/precipitation process; further reductions in concentration would be expected as a result of activated carbon filtration and reverse osmosis. Resulting concentrations in the effluent to the evaporation unit are shown in Table C-13.

C.2.2.2 Contaminant Emission Rates

Equations for predicting contaminant emission rates were provided by the engineering consultants to OWMC (Monenco 1987a). The separate approaches developed for organic chemicals and metals are discussed in the following subsections. A summary of the predicted evaporator emission rates is provided at the end of the section.

Organic Emission Rates. The following formula was used for estimating evaporator stack emission rates for organic chemicals (Monenco 1987):

$$Q_i = \frac{(S)(C_i)}{3600(t)(\rho_s)}$$

Where:

Q_i = emission rate for chemical i, g/s

S = flow of stream containing chemical i, t/a

C_i = concentration of chemical i in the stream, mg/l

ρ_s = stream density, t/m^3

t = period of evaporator operation, hr/a

TABLE C-13

METAL CONCENTRATIONS IN THE EFFLUENT TO THE EVAPORATOR UNIT

Contaminant	Concentration (mg/l)
Arsenic	0.05
Cadmium	0.001
Chromium	0.05
Nickel	0.05

Source: Monenco 1987a

The following assumptions were made in estimating the chemical emission rates:

- ρ_s = 1 t/m^3 (Monenco 1987a)
- t = 8 400 hr/a (Monenco 1987a)

For the organic contaminants evaluated on a stream-by-stream basis, stream flows to the evaporator and chemical concentrations after polishing were used in the calculations.

For the constituents of "oil and grease", emission rates were based on the concentrations in Table C-12 and an assumed stream flow of approximately 55 000 t/a. As shown in Table C-14, this flow represents the total quantity of all UCD categories of either inorganic or aqueous organic waste containing significant quantities of "oil and grease" sent to the physical/chemical treatment plant. To ensure that the emission rates calculated using this approach did not exceed the total quantity available for release, a mass balance was performed. Using the mass balance, an emission rate was calculated assuming that the entire quantity of each "oil and grease" constituent sent to the physical/chemical treatment plant would be emitted from the evaporator stack:

$$Q_i = \frac{C_i\ (f_A O_A + f_I O_I) \times 10^6}{(365 \times 24 \times 3600)}$$

Where:

Q_i = emission rate for chemical i, g/s

TABLE C-14

CATEGORIES OF INORGANIC AND AQUEOUS ORGANIC WASTE CONTAINING SIGNIFICANT QUANTITIES OF "OIL AND GREASE"[1]

UCD Category[2]	Quantity Sent to Physical/ Chemical Treatment Facility[3] (t/a)
113	3 170
121	10 570
123	3 910
133	2 115
141	10 570
225	1 185
257	885
262	290
263	585
282	50
285	10
286	60
289	2 120
416	115
431	3 805
433	2 115
441	12 680
445	740
TOTAL	54 975

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[1] Excluding inorganic wastes sent directly to solidification.

[2] See Appendix B for a discussion of the UCD classification system.

[3] Quantities based on the waste profile (see Appendix B).

C_i = chemical concentration in "oil and grease", mg/l

f_A = prorating factor for organics in aqueous organic wastes

O_A = total quantity of "oil and grease" in the aqueous organic wastes sent to the physical/chemical treatment plant

f_I = prorating factor for organics in inorganic wastes

O_I = total quantity of "oil and grease" in the inorganic wastes sent to the physical/chemical treatment plant

The following assumptions were made in the analysis:

- f_A = 1.9 (see Section C.2.2.1)
- f_I = 5.2 (see Section C.2.2.1)
- O_A = 134.6 t/a (see Appendix B)
- O_I = 49.2 t/a (see Appendix B)

The lower of the emission rates calculated using the two approaches were used in the risk assessment for constituents of "oil and grease".

Metal Emission Rates. The following formula was used to estimate the metal emission rates (Monenco 1987a):

$$Q_i = \frac{(S)(C_i)(C_d)}{3600\ (t)(\rho_s)}$$

Where:

Q_i = emission rate for metal i, g/s

S = flow of stream containing metal i, t/a

C_i = concentration of metal i in the stream, mg/l (see Table C-13)

C_d = concentration of water droplets in the evaporator plume, t/t

ρ_s = stream density, t/m^3

t = period of evaporator operation, hr/a

The following assumptions were made in performing the calculations:

- C_d = 25 x 10^{-6} (Monenco 1987a)
- ρ_s = 1 t/m^3 (Monenco 1987a)
- t = 8 400 hr (Monenco 1987a)
- S = 80 000 t/a (Monenco 1987a)

The value for C_d was based on data provided by the suppliers of evaporation equipment indicating that the concentration of water droplets in the evaporator plume after demisting will be less than 25 parts per million. The value for S represents the entire quantity of inorganic waste received at the physical/chemical treatment plant.

Emission rate estimates. Emission rate estimates for organics and metals were developed using the approaches outlined in this section. These estimates are presented in Table C-15.

C.2.3 FUGITIVE RELEASES

Fugitive releases may occur at various locations at the proposed OWMC facility. The fugitive emission estimates used

TABLE C-15

EVAPORATOR EMISSION RATES

(150 000 t/a facility)

Contaminant	Rate (g/s)
Acrylonitrile	1.4×10^{-4}
Benzene	2.7×10^{-5}
Benzo(a)anthracene	2.6×10^{-8}
Benzo(a)pyrene	7.3×10^{-8}
Butadiene	6.8×10^{-6}
Dichlorodifluoromethane	1.8×10^{-3}
Formaldehyde	6.5×10^{-7}
Naphthalene	1.8×10^{-3}
PCBs	2.7×10^{-8}
Phenol	2.0×10^{-5}
Styrene	2.6×10^{-5}
Tetrachloroethylene	9.1×10^{-5}
Toluene	4.8×10^{-4}
1,1,1-Trichloroethane	1.8×10^{-3}
Trichloroethylene	4.6×10^{-5}
Trichlorotrifluoroethane	1.8×10^{-3}
Xylene	9.1×10^{-5}
Arsenic	3.3×10^{-9}
Cadmium	6.6×10^{-11}
Chromium	3.3×10^{-9}
Nickel	3.3×10^{-9}

in the Phase 4B risk assessment were based on calculations performed by the engineering consultants to OWMC (Monenco 1987, Monenco 1987). Since these calculations were based on a single component system, i.e., the release of a pure chemical, it was necessary to extend the formulas developed by the engineering consultant to address the waste mixtures expected at the OWMC facility.

Empirical correlations capable of rigorously predicting vapor-liquid equilibria for non-ideal multi-component hydrocarbon mixtures are currently unavailable. Raoult's Law for ideal vapor-liquid equilibria was therefore used to approximate the behavior of components within a hazardous waste mixture. Raoult's Law is based on the following assumptions:

- The gas phase behaves as an ideal gas.
- The liquid phase behaves as an ideal solution.

Considering the uncertainty with which the composition of the waste streams to be received at the OWMC facility is known, the error associated with the use of these assumptions in estimating fugitive emission rates is believed to be relatively small.

Raoult's Law can be expressed mathematically as:

$$y_i = \frac{x_i P_i}{P_o}$$

Where:

y_i = mole fraction of component i in the gas phase

x_i = mole fraction of component i in the liquid phase

P_i = vapor pressure of component i, mm Hg

P_o = ambient pressure, mm Hg

Calculation of the mole fraction of a chemical in a particular waste requires: (a) the mass fraction of each chemical in the waste; and (b) the molecular weight of the waste as a whole. The mass fractions for each chemical in the organic and aqueous organic wastes were calculated in Appendix B. Given the variable composition of wastes to be received at the OWMC facility, however, it is difficult to determine the molecular weight of the waste mixture with great accuracy. Based on a consideration of the range of constituents expected to be present in the waste streams to be received at the OWMC facility, an average molecular weight for the components of the mixture other than the constituent of concern was assumed to be 150 g/mole. The mole fraction for a given chemical can therefore be estimated using the following relationship:

$$x = \frac{MS_i/MW_i}{(MS_i/MW_i + (1 - MS_i)/150)}$$

Where:

x = mole fraction of constituent, mol/mol

MS_i = mass fraction of the constituent, g/g

MW_i = molecular weight of the constituent, g/mol

The mole fractions calculated for surrogate chemicals using this approach are presented in Table C-16. (Mass fractions used in the calculations are reported in Appendix B.) Other chemical-specific data required in the estimation of fugitive emission rates are summarized in Table C-17.

The methodologies employed in calculating fugitive emissions from the various sources at the proposed OWMC facility are presented in the following subsections. A summary of the fugitive emissions from the various sources is provided at the conclusion of this section.

C.2.3.1 Bulk Waste Unloading and Transfer

Some waste arriving at the facility will be unloaded into below-ground reception basins (unloading tanks). Other wastes will be transferred directly to storage through a hose connection. For the initial plant throughput of 150 000 t/a, there will be thirteen tank truck unloading stations; nine for receiving inorganic wastes and four for organic wastes. (Additional stations will be added when the plant is expanded to a throughput of 300 000 t/a). A below-ground unloading tank equipped with a removable screened cage will be provided for each of the inorganic and two of the organic unloading stations for separating gross solids.

TABLE C-16

MOLE FRACTIONS OF SURROGATE CHEMICALS FOR FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	ORGANIC MOLE FRAC	AQUEOUS ORGANIC MOLE FRAC
ACETONE	8.56E-02	
ACRYLONITRILE	6.50E-04	8.15E-03
BENZENE	4.79E-03	4.99E-04
BENZO(A)ANTHRACENE	1.12E-05	2.23E-05
BENZO(A)PYRENE	4.04E-06	8.32E-06
BROMODICHLOROMETHANE		
BROMOFORM		
BUTADIENE,1,3-	5.55E-05	8.04E-04
CARBON TETRACHLORIDE	1.07E-03	
CHLOROBENZENE		
CHLOROFORM	2.39E-03	
DICHLORODIFLUOROMETHANE		6.82E-04
DIOXIN,2,3,7,8-EQ(EPA)		
DIOXIN,2,3,7,8-EQ(MOE)		
ETHANOL	1.54E-01	
ETHYLENE GLYCOL	1.48E-01	
ETHYLENE OXIDE	5.79E-05	
FORMALDEHYDE	3.04E-03	1.09E-02
HEXACHLOROBENZENE	5.11E-06	
HYDRAZINE	2.62E-07	
METHYL ETHYL KETONE	2.02E-02	
METHYLENE CHLORIDE	3.31E-02	
NAPHTHALENE		7.96E-04
NITROPHENOL,o-		
PCB	1.10E-03	1.96E-05
PETROLEUM DISTILLATES	6.75E-02	2.42E-02
PHENOL		2.33E-01
PYRIDINE	2.08E-03	
STYRENE		4.17E-03
TETRACHLOROETHYLENE	2.63E-02	1.36E-03
TETRAETHYL LEAD	2.09E-06	
TOLUENE	8.04E-02	4.99E-01
TRICHLOROETHANE,1,1,1-	2.47E-02	3.37E-03
TRICHLOROETHYLENE	2.84E-02	9.82E-04
TRICHLOROTRIFLUOROETHANE		8.81E-04
XYLENE	3.63E-02	4.52E-03

TABLE C-17

DATA INPUTS FOR FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	MOLEC WEIGHT	DIFF IN AIR @ 25 C (cm^2/sec)	Reference	Sc^-0.67	Kg (m/s)
ACETONE	58.08	1.06E-01	Shen 1980	0.7	3.59E-03
ACRYLONITRILE	53.064	1.04E-01	Lugg 1968	0.7	3.59E-03
BENZENE	78.114	9.32E-02	Lugg 1968	0.7	3.59E-03
BENZO(A)ANTHRACENE	228.3	5.30E-02	Calculated	0.5	2.56E-03
BENZO(A)PYRENE	252.3	5.16E-02	Calculated	0.5	2.56E-03
BROMODICHLOROMETHANE	163.83				
BROMOFORM	252.77				
BUTADIENE,1,3-	54.09	3.19E-02	Calculated	0.7	3.59E-03
CARBON TETRACHLORIDE	153.82	8.28E-02	Lugg 1968	0.6	3.08E-03
CHLOROBENZENE	112.56				
CHLOROFORM	119.378	8.88E-02	Lugg 1968	0.6	3.08E-03
DICHLORODIFLUOROMETHANE	120.92	9.40E-02	Calculated	0.6	3.08E-03
DIOXIN,2,3,7,8-EQ(EPA)	322				
DIOXIN,2,3,7,8-EQ(MCL)	322				
ETHANOL	46.069	1.19E-01	Lugg 1968	0.7	3.59E-03
ETHYLENE GLYCOL	62.07	1.01E-01	Calculated	0.7	3.59E-03
ETHYLENE OXIDE	44.05	1.31E-02	Calculated	0.7	3.59E-03
FORMALDEHYDE	30.026	1.70E-01	Calculated	0.7	3.59E-03
HEXACHLOROBENZENE	284.79	5.90E-02	Calculated	0.5	2.56E-03
HYDRAZINE	32.05	1.90E-01	Calculated	0.7	3.59E-03
METHYL ETHYL KETONE	72.1	9.03E-02	Lugg 1968	0.7	3.59E-03
METHYLENE CHLORIDE	84.933	1.04E-01	Lugg 1968	0.7	3.59E-03
NAPHTHALENE	128.2	7.00E-02	Calculated	0.6	3.08E-03
NITROPHENOL,O-	139.1				
PCB	328.4	4.00E-02	Calculated	0.5	2.56E-03
PETROLEUM DISTILLATES	130	7.57E-02	Calculated	0.6	3.08E-03
PHENOL	94.11	8.42E-02	Shen 1980	0.7	3.59E-03
PYRIDINE	79.102	9.50E-02	Calculated	0.7	3.59E-03
STYRENE	104.16	7.04E-02	Shen 1980	0.6	3.08E-03
TETRACHLOROETHYLENE	165.834	7.97E-02	Lugg 1968	0.6	3.08E-03
TETRAETHYL LEAD	323.44	1.00E-02	Calculated	0.5	2.56E-03
TOLUENE	92.141	8.49E-02	Lugg 1968	0.7	3.59E-03
TRICHLOROETHANE,1,1,1-	133.405	7.94E-02	Lugg 1968	0.6	3.08E-03
TRICHLOROETHYLENE	131.389	8.75E-02	Lugg 1968	0.6	3.08E-03
TRICHLOROTRIFLUOROETHANE	187.38	7.49E-02	Calculated	0.6	3.08E-03
XYLENE	106.16	6.91E-02	Lugg 1968	0.6	3.08E-03

Continued . . .

TABLE C-17 (continued)

DATA INPUTS FOR FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	VAPOR PRESS @ 10 C (mm Hg)	Reference	VAPOR PRESS @ 25 C (mm Hg)	Reference	VAPOR PRESS @ 70 C (mm Hg)	Reference
ACETONE	1.00E+02	CRC 1984	2.34E+02	Verscheuren 1983	1.18E+03	Reid 1977
ACRYLONITRILE	5.07E+01	Reid 1977	1.00E+02	Verscheuren 1983	5.97E+02	Reid 1977
BENZENE	4.52E+01	Reid 1977	9.45E+01	Reid 1977	5.48E+02	Reid 1977
BENZO(A)ANTHRACENE	7.83E-12	Analogy to BaP	5.00E-09	Mabey 1982	1.49E-05	Analogy to BaP
BENZO(A)PYRENE	7.83E-12	Calculated	5.60E-09	Mabey 1982	1.49E-05	Calculated
BROMODICHLOROMETHANE						
BROMOFORM						
BUTADIENE,1,3-	1.28E+03	Reid 1977	2.10E+03	Reid 1977	6.85E+03	Reid 1977
CARBON TETRACHLORIDE	5.60E+01	Reid 1977	1.13E+02	Verscheuren 1983	6.14E+02	Reid 1977
CHLOROBENZENE						
CHLOROFORM	9.82E+01	Reid 1977	1.93E+02	Reid 1977	9.81E+02	Reid 1977
DICHLORODIFLUOROMETHANE	3.80E+03	CRC 1984	4.25E+03	Verscheuren 1983	1.52E+04	CRC 1984
DIOXIN,2,3,7,8-EQ(EPA)						
DIOXIN,2,3,7,8-EQ(MOE)						
ETHANOL	2.33E+01	Reid 1977	5.81E+01	Reid 1977	5.39E+02	Reid 1977
ETHYLENE GLYCOL	3.37E-02	Reid 1977	1.25E-01	Verscheuren 1983	5.00E+00	CRC 1984
ETHYLENE OXIDE	7.58E+02	Reid 1977	1.10E+03	Verscheuren 1983	5.23E+03	Reid 1977
FORMALDEHYDE	2.35E+03	Reid 1977	3.87E+03	Reid 1977	1.25E+04	Reid 1977
HEXACHLOROBENZENE	3.61E-07	Calculated	1.69E-05	Calculated	5.60E-03	Calculated
HYDRAZINE	5.40E+00	Reid 1977	1.60E+01	Verscheuren 1983	1.44E+02	Reid 1977
METHYL ETHYL KETONE	4.52E+01	Reid 1977	1.00E+02	CRC 1984	5.53E+02	Reid 1977
METHYLENE CHLORIDE	2.29E+02	Reid 1977	4.33E+02	Reid 1977	2.00E+03	Reid 1977
NAPHTHALENE	6.63E-02	Reid 1977	9.40E-02	Calculated	4.20E+00	Reid 1977
NITROPHENOL,O-						
PCB	3.63E-05	Reid 1977	7.71E-05	Mabey 1982	1.37E-01	Reid 1977
PETROLEUM DISTILLATES	1.78E+00	Reid 1977	4.00E+00	GE 1980	3.39E+00	Calculated
PHENOL	8.19E-02	Reid 1977	5.00E-01	Verscheuren 1983	1.00E+01	CRC,1984
PYRIDINE	8.50E+00	Reid 1977	2.00E+01	Reid 1977	1.66E+02	Reid 1977
STYRENE	2.32E+00	Reid 1977	6.12E+00	Reid 1977	6.04E+01	Reid 1977
TETRACHLOROETHYLENE	7.73E+00	Reid 1977	1.40E+01	Mabey 1982	1.42E+02	Reid 1977
TETRAETHYL LEAD	1.00E+00	Reid 1977	1.50E-01	Verscheuren 1983	1.00E+01	CRC 1984
TOLUENE	1.23E+01	Reid 1977	2.82E+01	Reid 1977	2.03E+02	Reid 1977
TRICHLOROETHANE,1,1,1-	1.02E+01	Reid 1977	1.00E+02	Verscheuren 1983	7.60E+02	CRC 1984
TRICHLOROETHYLENE	3.51E+01	Reid 1977	7.38E+01	Reid 1977	4.39E+02	Reid 1977
TRICHLOROTRIFLUOROETHANE	1.76E+02	Reid 1977	3.32E+02	Reid 1977	1.52E+03	Reid 1977
XYLENE	3.04E+00	Reid 1977	5.87E+00	Verscheuren 1983	7.19E+01	Reid 1977

For the purpose of calculating potential fugitive emissions, it was conservatively assumed that all wastes (except PCBs) will be received by the unloading tanks. The unloading tanks will be equipped with covers or doors and an extraction fan connected to a vent exhaust system. Each extraction fan will be designed to maintain a negative pressure in the tank when the doors are closed.

Exhaust from the organic system will be sent to the incinerator under normal conditions. When the incinerator is not in operation, the organic exhaust will be sent to a scrubbing unit. Exhaust from the inorganic vent system will be sent to a scrubber/adsorber during routine operations.

Two routine scenarios were considered:

- Organic waste reception - normal operating conditions
- Inorganic waste reception - normal operating conditions

Emission calculations were performed for normal operation of the scrubbing system assuming 90% removal of air pollutants (Monenco 1987). The calculations in this section are based on a capacity of 150 000 t/a which represents the initial capacity of the facility under normal operation. At the ultimate capacity of 300 000 t/a, emissions under normal operating conditions will be doubled.

Organic Waste Reception. Under normal operating conditions and a facility capacity of 150,000 t/a, the fugitive emission rate associated with organic waste unloading and transfer has been provided by Monenco (1987):

$$Q_{si} = \frac{514\ (x_i)(P_i)(M_i)(1-\eta)}{T}$$

Where:

Q_{si} = quantity of species i released per shipment, g/shipment

x_i = mole fraction of species, dimensionless

P_i = vapor pressure of species at 10°C, mm Hg

M_i = molecular weight of species, g/mol

η = fraction of contaminant removed via scrubbing/adsorption, 0.9

T = temperature, 283K

Assuming three shipments per day, 250 days per year, the average emission rate can be calculated using the following equation:

$$Q_i = 4.33 \times 10^{-6}\ (x_i)(P_i)(M_i)\ \text{g/s}$$

At a peak capacity of 300 000 t/a there will be 6 shipments/day so that the emission rate will become:

$$Q_i = 8.66 \times 10^{-6}\ (x_i)(P_i)(M_i)\ \text{g/s}$$

Inorganic Waste Reception. Semi-pumpable inorganic wastes will be received by underground unloading tanks through open hatches. The tanks will be flushed with water at the end of each day, thus, a particular load of waste could remain in a tank for a maximum of eight hours. Each tank will be provided with two ventilation systems: (1) a high velocity intake used during the time that the tank hatch is open; and (2) a continuous purge system for the period that the tank hatch is closed. Both systems will vent to wet scrubbers.

Releases during waste reception and unloading will include:

- Emissions of displaced air as the tank is being filled;
- Emissions from the falling waste as it enters the open hatch; and
- Emissions from the continuous purge stream.

Releases of two toxic inorganic gases, hydrogen cyanide (HCN) and hydrogen sulfide (H_2S), have been considered by the engineering consultants to OWMC (Monenco 1987). The following assumptions were made for illustration purposes:

- One hundred shipments of waste containing NaSH and $Zn(CN)_4$ solutions would be received by OWMC each year. The mole fraction of H_2S and HCN in these wastes would be 0.34 ppm and 3.6 ppm, respectively.
- The vapor concentrations for H_2S and HCN in equilibrium with these streams would be 0.173 g/m^3 and 0.0023 g/m^3, respectively.
- Adsorber efficiency would be 90 percent.
- Airflow through the continuous purge will be 0.03 m^3/s.
- 15 m^3 of waste would be received per shipment.
- Shipments of waste would be received 250 d/a.

Based on these assumptions, the following emission estimates were calculated for the illustrative scenario (Monenco 1987):

- Displaced Air

  $Q_{di} = (S)(Y_i)(N)(1-\eta$

  Where:

  Q_d = emission rate, g/a

  S = volume per shipment, 15 m^3

  Y_i = vapor concentration, contaminant i, g/m^3

  N = number of shipments per year, 100

  η = adsorber efficiency, 0.9

  Therefore,

  Q_d, H_2S = 26 g/a

  Q_d, HCN = 0.3 g/a

- Vaporization from Falling Waste

  $$Q_{vi} = \frac{274.6\ (M_i)(P_i)(N)(1-\eta)}{T}$$

  Where:

  Q_{vi} = emission rate, g/a

  M_i = molecular weight, contaminant i, g/mol

  P_i = partial pressure, mm Hg

  T = temperature, 283 K

  N = number of shipments per year, 100

  η = adsorber efficiency, 0.9

  The partial pressure, P_i, for H_2S was calculated as follows:

  $P_i = 760\ H_i\ X_i$

Where:

P_i = partial pressure, mm Hg

H_i = Henry's Law constant, 367 atm-m^3/mole

X_i = liquid mole fraction, 0.34 x 10^{-6} mole/m^3

Therefore,

P_{H_2S} = 94.8 x 10^{-3} mm Hg

The partial pressure, P_i, for HCN was calculated as follows:

$P_i = (P_v)(x_i)$

Where:

P_i = partial pressure, mm Hg

P_v = vapor pressure, 400 mm Hg

x_i = liquid mole fraction, 3.6 x 10^{-6} mol/m^3

Therefore,

P_{HCN} = 1.44 x 10^{-3} mm Hg

Using these values, the following emission rates were calculated:

Q_v,H_2S = 31 g/a

Q_v,HCN = 0.4 g/a

- Continuous Purge

$Q_{c,i} = 3600\ (Y_i)(A)(D)(t)(N)(1-\eta)$

Where:

$Q_{c,i}$ = emission rate, g/a

Y_i = vapor mass fraction, g/m^3

A = continuous air purge flow, 0.03 m^3/s

D = dilution of contaminant in purge air, 0.20

t = maximum period a shipment is in the tank, 8 hr

N = number of shipments per year, 100

η = adsorber efficiency, 0.9

Therefore,

$Q_c, H_2S = 300$ g/a

$Q_c, HCN = 4.0$ g/a

- Total

$$Q_t = (Q_c + Q_v + Q_d)/(365 \times 24 \times 3600)$$

Where:

Q_t = total emission rate, g/s

Q_c = continuous purge emission rate, g/a

Q_v = vaporization from falling waste emission rate, g/a

Q_d = displacement air emission rate, g/a

Therefore,

$Q_t, H_2S = 1.1 \times 10^{-5}$ g/s

$Q_t, HCN = 1.5 \times 10^{-7}$ g/s

These estimates were developed for illustration purposes only, since precise estimates of the amount of NaSH and $Zn(CN)_4$ wastes to be received are not available.

C.2.3.2 Routine Leaks and Spills

Routine leaks and spills can be expected at any industrial facility. In evaluating leaks and spills that may occur during normal operation of the proposed OWMC special waste facility, the following releases were examined:

- Routine spills during waste transfer operations;
- Liquid leaks from valves, pumps and other equipment;
- Vapor leaks from valves, pumps and other equipment.

The methods used in calculating the magnitude of these releases are presented in this section.

Transfer Operations. Fugitive emissions may occur while unloading and transferring waste during hose coupling/ uncoupling or other related transfer operations. The magnitude of these emissions was based on empirical data developed in a study of hazardous waste transportation spills in the United States (Abkowitz, Eiger, and Srinivasan 1984). Data were available from this study for the fraction of cargo lost during various stages in the transportation cycle without any indication of how the spilled chemical would subsequently be partitioned between air, surface water and ground water.

The fraction of each hazardous waste constituent released to the atmosphere was calculated based on an equation developed by Stiver and MacKay (1982):

$$Q_i = \frac{(Kg_i)(A)(P_i)(x_i)(M_i)}{(R)(T)}$$

Where:

Q_i = emission rate for species i, g/s

Kg_i = the gas mass transfer coefficient for species i, m/s

A = area of the spill, m^2

P_i = vapor pressure of species i at 10°C, mm Hg

x_i = mole fraction for species i, mol/mol

M_i = molecular weight of species i, g/mol

R = gas constant, 0.0624 mm-Hg m^3/mol K

T = temperature, 283 K

The total area of waste generated by routine spills (A) was estimated assuming daily cleanup:

$$A = \frac{(W)(fw)}{(b)(\rho)}$$

Where:

A = total spill area, m^2

W = daily quantity of organic waste transported by tanker, t/d

fw = fraction of waste released, g/g

ρ = average density of wastes, kg/l

b = waste film thickness, m

Based on the facility design and operations, the following assumptions were made to approximate the area of spilled wastes available for volatilization:

- At 150 000 t/a plant capacity, 14 825 t/a organic waste will be transported by tanker during 250 d/a (Monenco, 1987).
- The fraction of the waste released at terminal points, i.e. loading and unloading points, will be 8×10^{-6} at the OWMC facility (Abkowitz, Eiger, and Srinivasan 1984). For routine spills on-site, the fraction of

waste released at unloading points will be 4×10^{-6}. It should be noted that the engineering consultants to OWMC have estimated a total spillage rate of 0.41 kg/d for the 150 000 t/a facility, a value which is comparable to the rate assumed for this calculation (Monenco 1987).

- The spilled wastes will form a thin film assumed to have a thickness of approximately 0.001 m.
- Average waste density is approximately 1.0 kg/l.

Therefore,

$$A = \frac{(14{,}825\ t/a)(4 \times 10^{-6})(10^{3}\ kg/t)(10^{-3}\ m^{3}/l)}{(250\ d/a)(0.001\ m)(1.0\ kg/l)}$$

$$A = 0.24\ m^{2}$$

Use of this equation requires the calculation of a mass transfer coefficient for the chemical of concern, which can be estimated using the equation of MacKay and Matsugu (1973):

$$Kg = 0.0048\ (U)^{0.78}\ (d)^{-0.11}\ (Sc)^{-0.67}$$

Where:

Kg = mass transfer coefficient, m/s

U = windspeed, m/s

d = diameter of pool, m

Sc = Schmidt number, dimensionless

Schmidt numbers, which represent the effect of fluid friction on mass transfer, were approximated using the method of Shen (1982) based on molecular weight of the compound. Compounds with molecular weights less than 100 were assigned a $Sc^{-0.67}$ of 0.7, those with molecular weight from 100 to 200 were assigned a $Sc^{-0.67}$ of 0.6, and those with molecular weights greater than 200 were assigned a $Sc^{-0.67}$ of 0.5. Values of wind speed and pool diameter were selected for particular spill scenarios.

It is important to recognize that the gas mass transfer coefficient is applicable to the prediction of emission rates of particular chemicals from spills. Mass transfer of a chemical in a complex solution or mixture is a more complicated process, which may involve transport across several phase boundaries. Additionally, transport may be controlled by other materials present in the mixture. However, since these complexities are extremely difficult to quantify for a waste stream and are, in any event, likely to reduce the value of the mass transfer coefficient, it was considered an appropriate and conservative assumption to use the gas mass transfer coefficient for the constituent of concern.

The emission rate calculated in this manner is sensitive to environmental parameters, specifically wind speed, which affects the mass transfer coefficient (Kg) and ambient temperature. For example, the emission rate at 0°C will be approximately three times lower than at 30°C. A wind speed of 1 m/s was used to correspond to the atmospheric stability class F subsequently used to model atmospheric transport (see Gartner Lee Associates Limited, 1982 for discussion of atmospheric stability classes).

Since these losses were assumed to be associated with transportation spills that occur outdoors, the releases will be directly to the atmosphere without any contaminant removal in an adsorber unit.

Therefore, at 150,000 t/a plant capacity the emission rate for routine spills is given by:

$$Q_i = \frac{(0.24)(Kg_i)(P_i)(M_i)(x_i)(250\ d/a)}{(0.0624)(283)(365\ d/a)}$$

$$Q_i = 9.31 \times 10^{-3}\ Kg_i P_i M_i x_i\ g/s$$

At a peak capacity of 300 000 t/a the emission rate will be doubled to:

$$Q_i = 1.86 \times 10^{-2}\ (Kg_i)(P_i)(M_i)(x_i)\ g/s$$

To ensure that this emission rate does not exceed the available quantity of wastes, a mass balance was performed, and an emission rate calculated assuming that the quantity of wastes spilled per day would all be volatilized during the same day:

$$Q_i = (W)(f_w)(x_i)$$

Where:

W = daily quantity of organic waste transported by tanker, t/a

f_w = fraction of waste released

x_i = mass fraction of contaminant i, g/g

$$Q_i = \frac{(14\ 825\ t/a)(4 \times 10^{-6})(1\ 000\ kg/t)(1\ 000\ g/kg)(X_i)(250\ d/a)}{(250\ d/a)(86\ 400\ s/d) \qquad (365\ d/a)}$$

$$Q_i = 1.88 \times 10^{-3}\ x_i\ \text{g/s}$$

The lower of the two emission rates was used for estimating fugitive emissions resulting from transfer operations.

Equipment Liquid Leaks. Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate due to equipment liquid leakage was estimated by Monenco (1987):

$$Q_i = (0.39\ \text{kg/h})(1\ \text{h}/3\ 600\ \text{s})(1\ 000\ \text{g/kg})(x_i)$$
$$= 1.08 \times 10^{-1}\ x_i\ \text{g/s}$$

Where:

Q_i = emission rate of species i, g/s

x_i = mass fraction of species i, g/g

To ensure that this emission rate does not exceed the available quantity of wastes, a mass balance was performed, and an emission rate calculated assuming that the quantity of waste spilled per day would all be volatilized during the same day. The total area of waste generated by equipment liquid leakage was estimated to be:

$$A = \frac{(w)(fw)}{(b)(\rho)}$$

Where:

A = total spill area, m^2

w = daily quantity of organic waste transported by tanker, t/d

fw = fraction of waste released, g/g

ρ = average density of wastes, 1.0 kg/l

b = waste film thickness, 0.001 m

Based on facility design and operations, (w)(fw) was assumed to be equal to 0.39 kg/h (Monenco 1987). Therefore:

$$A = \frac{(0.39\ \text{kg/h})(10^{-3}\text{m}^3/\text{l})(24\ \text{h/d})}{(0.001\ \text{m})(1.0\ \text{kg/l})}$$

$$A = 9.36\ \text{m}^2$$

Using a model developed by Stiver and MacKay (1982), the emission rate at 150 000 t/a can then be given by:

$$Q_i = \frac{(9.36\ \text{m}^2)(Kg_i)(P_i)(x_i)(M_i)}{(283\ \text{K})(0.0624)}$$

$$Q_i = 0.53\ (Kg_i)(P_i)(x_i)(M_i)\ \text{g/s}$$

The lower of the two emission rates was then used for estimating fugitive emissions associated with equipment liquid leakage.

Equipment Vapor Leaks. Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate due to equipment vapor leaks has been estimated by Monenco (1987):

$$Q_i = 3.4 \times 10^{-7}\ (x_i)(P_i)(M_i)\ \text{g/s}$$

Where:

Q_i = emission rate of species i, g/s

x_i = mole fraction of species i in waste, mol/mol

P_i = vapor pressure of species i at 10 C, mm Hg

M_i = molecular weight of species i, g/mol

At 300 000 t/a, the emission rate is expected to double.

C.2.3.3 Drum Handling

Contaminants may be released to the atmosphere as the result of various drum handling operations at the OWMC facility. The following sources of release have been evaluated in the risk assessment:

- Open drums of organic waste, including the transfer of material from the drums;
- Drum heating, organic wastes;
- Open drums of inorganic waste, including the transfer of material from the drums.

The methods used to calculate the magnitude of fugitive emissions from each of these sources are presented in this section.

Open Drums of Organic Wastes. The drum storage and handling area for organic wastes will be designed to accept wastes arriving in drums up to 200 l in size. Drums will be opened by removing the lid or screwed filling hole plug, or by cutting a hole in the top of the drum. Subsequently, the contents will be transferred to a receiving tank.

Fugitive vapors generated from open drums will be collected by a ventilation system which exhausts the collected air through an activated carbon adsorber. Normal emission calculations assume an adsorber efficiency of 90 percent. Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for drum opening is given by Monenco (1987):

$$Q_i = \frac{(A)(D_{i,B})(P)(P_{i1} - P_{i2})(M_i)(1-\eta)}{(R)(T)(Z)(P_{B,m})}$$

Where:

Q_i = emission rate, g/s

A = liquid surface area of all drums handled daily, cm^2

$D_{i,B}$ = diffusivity of species i in air @ 25°C, cm^2/s

P = ambient pressure, mm Hg

P_{i1} = partial pressure of species i @ gas-liquid interface, mm Hg

P_{i2} = partial pressure of species i, Z cm from surface of liquid, mm Hg

$P_{B,m}$ = logarithmic mean of the partial pressures of gases other than i at the waste-air interface and Z cm from the surface of liquid, mm Hg

R = gas constant, $(cm^3 \text{ mm Hg})/(g \text{ mol K})$

M_i = molecular weight of species i, g/mol

T = ambient temperature, K

x_i = mole fraction of i in wastes, mol/mol

Z = diffusion distance, cm

η = adsorber efficiency

P_i = vapor pressure of species i

Based on the facility design and operations (Monenco 1987), the following assumptions were made for calculation of emissions:

- A safety factor of 8 times the calculated diffusion rate was incorporated (Monenco 1987).
- 5 drums open continuously (including an allowance for wet drum lids and crushed drums) for 24 hours per day, 208 days per year.

A = 2 420 cm^2/drum

P = 760 mm Hg

P_{i2} = 0 mm Hg

$$P_{B,m} = \frac{(760-0) - (760-x_iP_i)}{\ln \dfrac{760-0}{(760-x_iP_i)}} = \frac{x_iP_i}{\ln\,[760/(760-x_iP_i)]}$$

R = 62 400(cm^3 mm Hg)/(g mol.K)

T = 298 K

Z = 5 cm

η = 0.9

Therefore, at 150 000 t/a plant capacity, on a daily average basis:

$$Q_i = \frac{8(2\ 420\text{cm}^2)(D_{i,B})(760\text{mm Hg})x_iP_iM_i(1-0.9)\ln[760/(760-x_iP_i)]}{62\ 400(\text{cm}^3\text{ mm Hg})/(\text{g mol K})\ (298\text{ K})\ (5\text{ cm})\ (x_iP_i)}$$

$$Q_i = 0.016\ (D_{i,B})(M_i)\ \ln\,[760/(760-x_iP_i)]\ \text{g/s per drum}$$

Assuming five drums opened per day, 208 days per year, the emissions can be calculated as:

$$Q_i = 4.5 \times 10^{-2}(D_{i,B})(M_i) \ \ln\,[760/(760-x_iP_i)] \text{ g/s}$$

At the peak capacity of 300 000 t/a the emission rate will be doubled so that:

$$Q_i = 1.14 \times 10^{-1}(D_{i,B})(M_i) \ \ln\,[760/(760-x_iP_i)] \text{ g/s}$$

Heating Drums of Organic Waste. An enclosed drum heating chamber will be provided at the OWMC facility for the warming of high viscosity organic waste. The heating of the waste would be expected to lead to increased volatile losses. Fugitive emissions from the drum heating chamber will be vented to the atmosphere through a carbon adsorption system. The emission rate for drum heating is given by (Monenco 1987):

$$Q_i = \frac{(A)(D_{1,B})(P)(P_{i1} - P_{i2})(M_i)(1-\eta)}{(R)(T)(Z)(P_{B,m})}$$

Where:

Q_i = emission rate, g/s

A = liquid surface area of all drums handled daily, cm^2

$D_{i,B}$ = diffusivity of species i in air @ 70°C, cm^2/s

P = ambient pressure, mm Hg

P_{i1} = partial pressure of species i @ gas-liquid interface, mm Hg

P_{i2} = partial pressure of species i, 5 cm from surface of liquid, mm Hg

$P_{B,m}$ = logarithmic mean of the partial pressures of gases other than i at the waste-air interface and 5 cm from the surface of liquid, mm Hg

R = gas constant, (cm^3 mm Hg)/(g mol K)

M_i = molecular weight of species i, g/mol

T = ambient temperature, K

x_i = mole fraction of i in wastes, mol/mol

Z = diffusion distance, cm

η = adsorber efficiency

P_i = vapor pressure of species i, mm Hg

Based on the facility design and operations (Monenco 1987), the following assumptions were made for calculation of emissions:

T = 70°C or 343 K

Z = 5 cm

A = 25 cm^2

η = 0.90

$D_{70^\circ} = D_{25^\circ} \times (343/298)^{1.5} = D_{25^\circ} \times 1.235$

P = 760 mm Hg

$P_{i,2}$ = 0 mm Hg

$$P_{B,m} = \frac{(760-0)-(760-x_iP_i)}{\ln\dfrac{(760-0)}{(760-x_iP_i)}} = \frac{x_iP_i}{\ln[760/(760-x_iP_i)]}$$

R = 62 400 (cm^3 mm Hg)/(g mol K)

Therefore, at 150 000 t/a plant capacity:

$$Q_{di} = \frac{(25\ \text{cm}^2)(1.235\ D_{i,B25})(760\ \text{mm Hg})(x_i P_i M_i)(1-0.9)\ln[760-x_i P_i]}{62\ 400(\text{cm}^3\ \text{mm Hg})\ /\ (\text{mole K})(343\ \text{K})(5)(x_i P_i)}$$

$$Q_{di} = 2.19 \times 10^{-5}(D_{i,B25})(M_i)\ln[760/760-x_i P_i)]\ \text{g/s/drum}$$

If it is assumed that 16 drums are heated at one time for a total of 208 days per year, then the emission rate can be calculated as:

$$Q_{di} = 2.00 \times 10^{-4}(D_{i,B25})(M_i)\ln[760/760-x_i P_i)]\ \text{g/s}$$

It should be noted that Monenco indicates that an 8-fold safety factor may be appropriate in calculating emissions resulting from drum heating. Although this factor has not been incorporated, it would not have a significant impact on overall fugitive emissions.

Handling Drums of Inorganic Waste. Drums containing inorganic wastes will be opened and tipped through one of four unloading stations into a chute connected to a below grade unloading tank. Fugitive emissions from the transfer operation are extracted and routed to the inorganic exhaust scrubber/adsorber system (Monenco 1987).

For purposes of estimating emissions, it was assumed in the risk assessment that an equivalent of ten open drums (which includes an allowance for wet drum lids and crushed drums) would be processed under normal operating conditions and a facility capacity of 150 000 t/a (Monenco 1987). The emission rate using hydrogen cyanide and hydrogen sulfide for illustration purposes can then be estimated as:

$$Q_{HCN} = (0.07\ g/d)(1\ d/24\ h)(1\ h/3\ 600\ s)(208\ d/a)/(365\ d/a)$$

$$= 4.62 \times 10^{-7}\ g/s$$

$$Q_{H_2S} = (1.8\ g/d)(1\ d/24\ h)(1\ h/3\ 600\ s)(208\ d/a)/(365\ d/a)$$

$$= 1.19 \times 10^{-5}\ g/s$$

At the 300 000 t/a capacity, 20 drums would be processed so that:

$$Q_{HCN} = 9.23 \times 10^{-7}\ g/s$$

$$Q_{H_2S} = 2.37 \times 10^{-5}\ g/s$$

These rates provide an illustration of expected HCN and H_2S emissions.

C.2.3.4 Waste Storage

Tanks will be used for storage, blending and pre-treatment of pumpable wastes received from off-site waste generators as well as from on-site sources. Fugitive emissions will result from the displacement of vapors from the storage tanks as they are being filled.

For purposes of the risk assessment, two scenarios were considered:

- organic waste storage - normal operating conditions (all organics)
- inorganic waste storage - normal operating conditions (hydrogen cyanide as surrogate chemical)

At 150 000 t/a capacity, under normal operating conditions, the displaced vapors from the organic waste storage will be vented to the incinerator, except for 1760 hours per year, when the incinerator is out of operation and the vapors will be vented through an activated carbon adsorber. The emission rates for fugitive emissions were calculated for a day when the incinerator is out of operation assuming an adsorber efficiency of 90%. At 300 000 t/a plant capacity, fugitive emissions will be eliminated by the presence of a second incinerator.

Organic Waste Storage. Under normal operating conditions, hydrocarbon emissions from organic waste storage will be routed directly to the incinerator. In the event that the incinerator is out of service, the gases will be vented to the atmosphere through an activated carbon adsorber. The emission rate for organic waste storage working losses for the 150 000 t/a facility has been given by Monenco (1987) as:

$$Q_i = \frac{(V)(x_i)(P_i)(M_i)(1-\eta)}{(R)(T)}$$

Where:

Q_i = emission rate, g/shipment

R = gas constant; 0.0624 (m^3 mm Hg)/(mol K)

T = temperature of tank, K

V = volume of shipment, m^3

x_i = mole fraction of species i in waste, mol/mol

P_i = vapor pressure of species i @ T, mm Hg

M_i = molecular weight of species i, g/mol

Based on the facility design and operations (Monenco 1987), the following assumptions were made for calculation of emissions:

- 100% saturation of air with species i is attained
- Volume of shipment is 15 m^3 ($V = 15\ m^3$)
- 90% efficiency of adsorber ($\eta = 0.9$)
- 7.5 shipments arrive per day, 250 days per year
- Incinerator unavailability = 1760 hours. Assuming waste is received 5 days per week and the plant is shut down for 2 weeks, then the time emissions will be occurring is 42.5 days.

T = 283 K

N_o = 7.5 shipments/d

R = 0.0624 (m^3 mm Hg)/(mol K)

Therefore, at 150 000 t/a plant capacity:

$$Q_i \quad \frac{(15\ m^3/\text{shipment})\ P_i x_i M_i\ (1-0.9)}{0.0624\ (m^3\ \text{mm Hg})/(\text{mol.K})(283\ K)}$$

$$Q_i = 8.49 \times 10^{-2}(x_i)(P_i)(M_i)\ \text{g/shipment}$$

Assuming that 7.5 shipments are received per day, and that emissions occur for a total of 42.5 days per year, then the emission per shipment can be converted to an emission rate as:

$$Q_i = 8.6 \times 10^{-7} (x_i)(P_i)(M_i) \text{ g/s}$$

At a peak capacity of 300 000 t/a, 15 shipments of waste are expected to be received each day. Although unexpected, if both incinerators are out of service at the same time, the emission rate is expected to double.

Inorganic Waste Storage. Under normal operating conditions and a plant capacity of 150 000 t/a, the emission rate for inorganic waste storage working losses is estimated to be (Monenco 1987):

$$Q_i = \frac{(P_o)(V)(M_i)(1-\eta)}{(R)(T)}$$

Where:

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flow rate of contaminant, m^3/s at standard conditions

R = gas constant, (m^3 mm Hg)/(mol K)

T = standard condition temperature, K

M_i = molecular weight of compound, g/mol

η = adsorber efficiency

Based on the facility design and operations (Monenco 1987), the following assumptions were made for illustration purposes:

- 1 600 t/a (approximately 1 500 m^3/a) of cyanide bearing waste is received

- The adsorber efficiency is 90% ($\eta = 0.9$)
- Mole fraction of HCN in the vapor in equilibrium with solution containing 1000 mg of CN per liter at pH 9.5 is 1.2×10^{-6} at 10°C
- Wastes are received 250 d/a

P_o = 760 mm Hg

V = $(1\ 500\ m^3/a)(1.2 \times 10^{-6})(1\ a/250\ d)(1\ d/86\ 400\ s)$

= $8.33 \times 10^{-11}\ m^3/s$

R = $0.0624\ (m^3\ mm\ Hg)/(mol\ K)$

T = 273 K

M_{HCN} = 27.03 g/mol

Therefore,

$$Q_{HCN} = \frac{(760\ mm\ Hg)(8.33 \times 10^{-11} m^3/s)(27.03\ g/mol)(1-0.9)(250\ d/a)}{0.0624\ (m^3\ mm\ Hg)/(mol\ K)\ (273\ K)(365\ d/a)}$$

$$Q_{HCN} = 6.88 \times 10^{-9}\ g/s$$

C.2.3.5 Physical/Chemical Treatment Plant

Aqueous organic and certain inorganic wastes will be treated in the physical/chemical plant. Potential fugitive emissions from the following physical/chemical treatment processes have been evaluated in the risk assessment:

- Dissolved Air Flotation (DAF) for the separation of hydrocarbons from aqueous phase;
- Emulsion breaking for the separation of hydrocarbons from the aqueous phase; and
- Oxidation/reduction reactions for the destruction of cyanides and reduction of chromium.

Estimates were made for fugitive emissions arising from these sources assuming 150 000 t/a plant capacity.

Dissolved Air Flotation Unit. The DAF unit will be used at the OWMC facility to separate suspended oil from aqueous solutions. The air above the liquid is first pressurized thereby increasing the solubility of air in the liquid. The pressure is then quickly released causing nucleation of air bubbles in the aqueous liquid which bubble to the surface. As the air bubbles rise through the waste liquid, globules of hydrocarbon adhere to the bubbles and are carried to the surface. The end result is that liquid hydrocarbon will tend to collect on the surface of the aqueous waste where, due to the action of the air, a layer of froth will be formed. The emission calculation assumes that the air is saturated with the hydrocarbon in the froth. This air is then released to the atmosphere through an activated carbon adsorber.

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for the DAF unit is given by (Monenco 1987):

$$Q_i = \text{(molar flow rate of air) (mole fraction of species in gas phase)}$$

$$= \frac{(P_s)(V)\ (x_i)(P_i)(M_i)(1-\eta)}{(R)(T)(P)}$$

Where:

Q_i = emission rate, g/s

P = ambient pressure, 760 mm Hg

R = gas constant, 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature, 273 K

x_i = mole fraction of species i in the oily waste (excluding water), mol/mol

P_i = vapor pressure of species i @ 25°C, mm Hg

M_i = molecular weight of species i, g/mol

V = volumetric flowrate of air at standard conditions, m^3/s

P_s = pressure at standard conditions, 760 mm Hg

η = adsorber efficiency

Based on the facility design and operations (Monenco 1987), the following assumptions were made for calculation of emissions:

- Air released can be assumed to be continuous based on 2.4 shifts/day average for 250 d/a
- The volumetric flowrate is 2.78×10^{-4} Nm^3/s for 2 units at 300 000 t/a, or 1.39×10^{-4} Nm^3/s at 150 000 t/a

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(760 \text{ mm Hg})(1.39 \times 10^{-4} \text{ Nm}^3/\text{s})\ x_i P_i M_i (1-0.9)}{0.0624\ (\text{m}^3 \text{ mm Hg})/(\text{mol K})\ (273 \text{ K})(760 \text{ mm Hg})}$$

$$Q_i = 8.15 \times 10^{-7}(x_i)(P_i)(M_i) \text{ g/s}$$

Based on 2.4 shifts per day, 8 hours per shift, 250 days per year, the annual average emission rate can be calculated as:

$$Q_i = 4.47 \times 10^{-7}(x_i)(P_i)(M_i) \text{ g/s}$$

At the peak capacity of 300 000 t/a the emissions would double to:

$$Q_i = 8.93 \times 10^{-7}(x_i)(P_i)(M_i) \text{ g/s}$$

Emulsion Breaking. Oil (hydrocarbon)/water emulsions in the aqueous organic wastes will be broken by heating. As the emulsion-breaking tank is filled, vapor will be displaced and vented through an adsorber/scrubber system. Air in equilibrium with hydrocarbon/oil will be vented through an adsorber which is assumed to have an efficiency of 90%. The following emission calculations are based on a 150 000 t/a plant capacity. The estimates would double at the ultimate capacity of 300 000 t/a.

Under normal operating conditions and a facility capacity of 150 000 t/a, the emission rate for the emulsion-breaker is given by Monenco (1987):

$$Q_i = \frac{(P_o)(V)\ (x_i)(P_i)(M_i)(1-h)}{(R)(T)(P_o)} = \frac{(V)(x_i)(P_i)(M_i)(1-\eta)}{(R)(T)}$$

Where:

Q_i = emission rate, g/s

P_o = pressure of system, 760 mm Hg

T = temperature of gas, 298 K

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

x_i = mole fraction of species i in the oily waste, excluding water, mol/mol

P_i = vapor pressure of species i at T, mm Hg

M_i = molecular weight of species i, g/mol

V = volume expelled per second, m^3

Based on the facility design and operations (Monenco 1987), the following assumptions were made for calculation of emissions:

- 3.5 batches per day each of 25 m^3 for 250 d/a
- 90% adsorber efficiency (η=0.9)

Thus, the emission rate at 150 000 t/a can be calculated as:

$$Q_i = \frac{(25\ m^3/\text{batch})\ M_i x_i P_i\ (1-0.9)}{0.0624\ (m^3\ \text{mm Hg})/(\text{mol K})(298\ K)}$$

$$Q_i = 1.34 \times 10^{-1}(x_i)(P_i)(M_i)\ \text{g/batch}$$

Based on 3.5 batches per day for 250 days per year, the emission rate can be calculated as:

$$Q_i = 3.70 \times 10^{-6}\ (x_i)(P_i)(M_i)\ \text{g/s}$$

At the peak capacity of 300 000 t/a the emissions would double to:

$$Q_i = 7.40 \times 10^{-6}\ (x_i)(P_i)(M_i)\ \text{g/s}$$

Oxidation/Reduction of Cyanide Wastes. The cyanide bearing waste will be destroyed by the addition of an oxidizing agent such as hypochlorite or hydrogen peroxide. Air from the unit will be vented through the scrubber/adsorber. For calculation of emissions under normal operating conditions, it was assumed that the hydrogen cyanide concentration in air leaving the unit will be in equilibrium with the concentration in solution. This air will be vented through a scrubber with an efficiency of 90 percent.

Under normal operating conditions and a facility capacity of 150 000 t/a, the hydrogen cyanide emission rate is given by (Monenco 1987):

$$Q_i = \frac{(P_o)(V)(M_{HCN})(1-\eta)}{(R)(T)}$$

Where:

Q_i = emission rate, g/s

P_o = standard condition pressure, mm Hg

V = volumetric flowrate at standard condition, m^3/s

R = gas constant = 0.0624 (m^3 mm Hg)/(mol K)

T = standard condition temperature, 273 K

η = adsorber efficiency

M_{HCN} = molecular weight of HCN, g/mol

Based on facility design and operations (Monenco 1987), the following assumptions were made for illustration purposes:

- 1 100 t/a cyanide bearing waste at 1% by weight CN^- = 11 t/a of CN^- at 1 000 mg/L of CN^-; total volume to be treated = 11 000 m^3
- 275 batches of 40 m^3 each are treated per year (250 d/a)
- air flow = 0.5 m^3/minute
- average mole fraction of HCN in the vapor over 6 hour period = 3.9×10^{-6}

- 0.0007 m^3 of HCN vapor evolved per batch at 60°C or 0.00058 Nm^3/batch ($V = 0.00058\ Nm^3$/batch)
- scrubber/adsorber efficiency is 90% ($\eta = 0.9$)
- $M_{HCN} = 27.03$ g/mol
- $P = 760$ mm Hg

Therefore, at 150 000 t/a:

$$Q_i = \frac{(760\ \text{mm Hg})(5.8 \times 10^{-4} Nm^3/\text{batch})(27.03\ \text{g/mol})(1-0.9)}{0.0624\ (m^3\ \text{mm Hg})/(\text{mol K})(273\text{K})}$$

$$Q_i = 0.07\ \text{g HCN/batch}\ (275\ \text{batches/a})(1\ \text{a}/365\ \text{d})(1\ \text{d}/86\ 400\ \text{s})$$

$$= 6 \times 10^{-7}\ \text{g/s HCN}$$

At the peak capacity of 300 000 t/a the emissions would double to 1.8×10^{-6} g/s HCN.

C.2.3.6 Summary of Emission Estimates

Contaminant emission rates associated with the fugitive release sources considered in the risk assessment calculated using the approaches described in this section are summarized in Table C-18.

TABLE C-18

PROJECTED FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	PHYS/CHEM FUGITIVE EMISSIONS (g/sec)	OTHER FUGITIVE EMISSIONS (g/sec)	TOTAL FUGITIVE EMISSIONS (g/sec)
ACETONE	0.00E+00	1.42E-02	1.42E-02
ACRYLONITRILE	1.80E-04	5.71E-05	2.37E-04
BENZENE	1.53E-05	5.68E-04	5.84E-04
BENZO(A)ANTHRACENE	1.06E-16	5.96E-16	7.02E-16
BENZO(A)PYRENE	4.88E-17	2.34E-16	2.83E-16
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	3.78E-04	3.55E-05	4.13E-04
CARBON TETRACHLORIDE	0.00E+00	2.66E-04	2.66E-04
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	6.60E-04	6.60E-04
DICHLORODIFLUOROMETHANE	1.45E-03	0.00E+00	1.45E-03
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	9.79E-03	9.79E-03
ETHYLENE GLYCOL	0.00E+00	6.09E-04	6.09E-04
ETHYLENE OXIDE	0.00E+00	2.60E-06	2.60E-06
FORMALDEHYDE	5.19E-03	4.84E-03	1.00E-02
HEXACHLOROBENZENE	0.00E+00	9.41E-13	9.41E-13
HYDRAZINE	0.00E+00	7.98E-09	7.98E-09
METHYL ETHYL KETONE	0.00E+00	2.24E-03	2.24E-03
METHYLENE CHLORIDE	0.00E+00	1.34E-02	1.34E-02
NAPHTHALENE	3.98E-08	0.00E+00	3.98E-08
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00
PCB	2.06E-12	1.77E-08	1.77E-08
PETROLEUM DISTILLATES	5.21E-05	6.73E-03	6.78E-03
PHENOL	4.55E-05	0.00E+00	4.55E-05
PYRIDINE	0.00E+00	1.48E-04	1.48E-04
STYRENE	1.10E-05	0.00E+00	1.10E-05
TETRACHLOROETHYLENE	1.31E-05	3.67E-03	3.69E-03
TETRAETHYL LEAD	0.00E+00	4.98E-07	4.98E-07
TOLUENE	5.39E-03	7.20E-03	1.26E-02
TRICHLOROETHANE,1,1,1-	1.87E-04	4.21E-03	4.39E-03
TRICHLOROETHYLENE	3.95E-05	4.94E-03	4.98E-03
TRICHLOROTRIFLUOROETHANE	2.27E-04	0.00E+00	2.27E-04
XYLENE	1.15E-05	3.02E-03	3.03E-03
HYDROGEN CYANIDE	6.00E-07	6.28E-07	1.23E-06
HYDROGEN SULFIDE	0.00E+00	2.29E-05	2.29E-05

C.3 RELEASES FROM THE ENGINEERED LANDFILL

The proposed OWMC facility will include an engineered landfill for the disposal of solid residues produced by various on-site treatment processes (Monenco 1987). These residues will consist of:

- Approximately 2 700 t/a of slag (a fused material composed primarily of metal compounds) generated in the rotary kiln incinerator;
- Approximately 12 000 t/a of solidified material from an incinerator solidification unit. This solidification unit will receive salt and ash from the waste heat boiler, spray dryer, and electrostatic precipitator components of the incineration system. The salt and ash will be mixed with various reagents (e.g., Portland cement and lime) to produce a solid material.
- Approximately 99 000 t/a of treated residues from the main solidification plant. The main solidification plant will receive solid inorganic wastes from off-site generators and solid residues generated in the physical/chemical treatment plant. These wastes will be mixed with various reagents (e.g., Portland cement, sodium silicate solution, and lime) to produce a solid material.

Each of these three types of waste will be composed primarily of inorganic constituents, such as silica and metals, although small amounts of organic chemicals may also be present.

The purpose of the landfill is to contain potentially hazardous chemicals that may be present in the incinerator residues and solidified treated residues. However, in practice the compacted clay cover placed on the landfill will allow the infiltration of some small fraction of the incident rainfall or snowmelt. In addition, water may seep into the solidified wastes from the bottom or sides of the landfill. As this water percolates through the landfill, chemicals will dissolve out of the solidified, treated residues to generate an aqueous liquid known as leachate. Subsequent migration of leachate from the landfill, either laterally to surface water or downward to ground water, may result in contamination of the off-site environment.

The first step in assessing the potential public health risks associated with releases from the OWMC landfill was to estimate of the contaminant concentrations in the leachate. This process consisted of two steps:

- Characterization of the chemical composition of the wastes to be deposited in the OWMC landfill; and
- Estimation of the release of specific chemicals from the landfilled materials to the leachate.

The approaches used in these two steps are described in this Appendix.

C.3.1 CHARACTERIZATION OF LANDFILLED MATERIALS

C.3.1.1 Incinerator Slag

Information on the constituents present in slag produced by existing hazardous waste incinerators has been compiled by the engineering consultants to OWMC (Monenco 1987). These data were generated at three incinerators (Kommunekemi in Denmark, HIM Biebesheim in Germany, and SAKAB in Sweden) which are similar in design and operation to the proposed OWMC facility. These data were considered to represent the "typical" composition of slag for the purposes of the risk assessment. Since the chemical composition of slag may vary significantly depending on the characteristics of the waste being incinerated, the highest concentration reported for each contaminant at the three facilities was used in the assessment. These concentrations are presented in Table C-19.

The inorganics reported in the incinerator slag from the Kommunekemi, HIM, and SAKAB facilities were categorized into three groups:

- Potentially hazardous inorganics for which available toxicity data are sufficient for the purposes of risk assessment. These contaminants include cadmium, lead, and chromium.
- Potentially hazardous inorganics for which available toxicity data are insufficient for the purposes of risk assessment. These contaminants include aluminum, manganese, cobalt, and boron.

TABLE C-19

CONTAMINANT CONCENTRATIONS IN INCINERATOR SLAG

Metal	Maximum Concentration (ppm)
Iron	764 400
Silica	413 700
Titanium	300 000
Aluminum	119 400
Calcium	80 000
Sodium	50 000
Magnesium	30 000
Barium	38 000
Copper	30 000
Potassium	5 300
Manganese	5 000
Chromium	39 400
Strontium	2 000
Molybdenum	2 000
Nickel	2 000
Tin	500
Zinc	1 000
Cobalt	43 000
Lead	2 000
Boron	50
Zirconium	300
Silver	10
Vanadium	100
Palladium	20 500

Derived from Monenco 1987.

- Inorganics, such as iron, sodium, and potassium, that pose relatively low public health hazards.

To allow for the absence of adequate toxicity data for a portion of the potentially hazardous material, inorganics for which sufficient toxicity data are available were selected as surrogates. These surrogates are denoted in Table C-20. The quantities of those substances which may be hazardous, but for which adequate toxicity data are not available, were represented by these surrogates in the risk assessment.

As shown in Table C-20, approximately 30 percent of the total quantity of metals in incinerator slag was considered to pose a potential human health hazard. Toxicity data characterizing approximately 11 percent of the total metal quantity was available. The mass fraction of each surrogate in the slag was therefore calculated as follows:

$$C_{s,i} = \frac{(C_i)\ (Q_T)}{Q_D}$$

Where:

$C_{s,i}$ = estimated concentration for surrogate i in incinerator slag, ppm

C_i = maximum reported concentration for contaminant i in incinerator slag, ppm

Q_T = percentage of the total quantity of metal considered to be potentially hazardous, 30.5%

Q_D = percentage of the total quantity of metals for which sufficient toxicity data are available, 11.2%

TABLE C-20

CATEGORIZATION OF METALS IN SLAG

Metal	Potentially Hazardous in Slag	Surrogate (Toxicity Data Available)
Iron	No	--
Silica	No	--
Titanium	No	--
Aluminum	Yes	No
Calcium	No	--
Sodium	No	--
Magnesium	No	--
Barium	Yes	Yes
Copper	Yes	Yes
Potassium	No	--
Manganese	Yes	No
Chromium	Yes	Yes
Strontium	Yes	No
Molybdenum	Yes	No
Nickel	Yes	Yes
Tin	Yes	No
Zinc	Yes	Yes
Cobalt	Yes	No
Lead	Yes	Yes
Boron	Yes	No
Zirconium	No	No
Silver	Yes	Yes
Vanadium	Yes	No
Palladium	Yes	No

The concentrations of surrogate metals in slag calculated using this approach are presented in Table C-21.

C.3.1.2 Incinerator Salt and Ash

The concentrations of certain potentially hazardous constituents of incinerator salt and ash have also been reported by the engineering consultants to OWMC (Monenco 1987). These data, gathered at the HIM Biebesheim Incineratory in West Germany, are shown in Table C-22.

The surrogate approach described in Section C.3.1.1 was also applied to characterize the metal content of salt and ash. The surrogates selected are denoted in Table C-23. Concentrations for the surrogates in the salt and ash were calculated using the following equation:

$$C_{s,i} = \frac{(C_i)\ (Q_T)}{Q_D}$$

Where:

$C_{s,i}$ = estimated concentration for surrogate i in incinerator salt and ash, ppm

C_i = reported concentration for contaminant i in incinerator salt and ash, ppm

Q_T = percentage of the total quantity of metal considered to be hazardous, 2.1%

Q_D = percentage of the total quantity of metal considered to be hazardous, 1.6%

The concentrations of surrogate metals in salt and ash calculated using this approach are presented in Table C-24.

TABLE C-21

CONCENTRATIONS OF SURROGATE CHEMICALS IN INCINERATOR SLAG

Contaminant	Concentration (ppm)[1]	Quantity (t/a)[2]
Barium	103 090	278
Copper	81 390	220
Chromium	106 890	289
Nickel	5 430	15
Zinc	2 713	7.3
Lead	5 430	15
Silver	30	0.07

---

[1] Derived from data reported in Monenco (1987, 1986d)

[2] Based on 2 700 t/a slag generated by the rotary kiln incinerator (Monenco 1986d, 1987).

TABLE C-22

CONTAMINANT CONCENTRATIONS IN INCINERATOR SALT AND ASH

Metal	Concentration (ppm)
Aluminum	2 800
Barium	295
Boron	710
Cadmium	260
Calcium	130 000
Chromium	160
Copper	840
Iron	10 600
Lead	5 050
Magnesium	3 000
Manganese	160
Mercury	14
Nickel	135
Potassium	11 900
Sodium	150 000
Tin	840
Titanium	1 050
Vanadium	49
Zinc	9 300

Source: Monenco 1987.

TABLE C-23

CATEGORIZATION OF METALS IN INCINERATOR SALT AND ASH

Metal	Potentially Hazardous in Salt or Ash	Surrogate (Toxicity Data Available)
Aluminum	Yes	No
Barium	Yes	Yes
Boron	Yes	No
Cadmium	Yes	Yes
Calcium	No	--
Chromium	Yes	Yes
Copper	Yes	Yes
Iron	No	--
Lead	Yes	Yes
Magnesium	No	--
Manganese	Yes	No
Mercury	Yes	Yes
Nickel	Yes	Yes
Potassium	No	--
Sodium	No	--
Tin	Yes	No
Titanium	No	--
Vanadium	Yes	No
Zinc	Yes	Yes

TABLE C-24

CONCENTRATIONS OF SURROGATE CHEMICALS IN INCINERATOR SALT AND ASH

Contaminant	Concentration (ppm)[1]	Quantity (t/a)[3]
Cadmium	330	2.2
Mercury	20	0.1
Barium	380	2.5
Copper	1 080	7.0
Chromium	210	1.3
Nickel	170	1.1
Zinc	11 940	77.6
Lead	6 480	42.1
Nitrate	40	0.26
PCDD/PCDF[2]	0.041	2.7×10^{-4}

---

[1] Data for inorganic contaminants derived from Monenco (1987); estimate of PCDD/PCDF concentration developed by ENVIRON (see Section C.3.1.2).

[2] Polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) expressed as 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents (TEQ). See Appendix H for a discussion of TEQ factors.

[3] Based on 6 500 t/a salt and ash generated by the rotary kiln incinerator (Monenco 1987).

The concentration of nitrate in the salt and ash is also included in Table C-24; the data for this compound were compiled by the engineering consultants to OWMC (Monenco 1987).

Data provided by the engineering consultants to OWMC indicates that it is likely that a small portion (less than 1 percent) of the salt and ash produced by the incinerator will consist of organic material (Monenco 1986d). These data, however, do not identify specific organic chemicals that may be expected. It is known, however, that salt and ash collected in the boiler and air pollution control equipment may contain hazardous organics that condense onto the particulates in the post-combustion zone of the incinerator.

Of the organics that may be present in incinerator salt and ash, public health concerns have focused primarily on a family of compounds known as polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) since they are generally believed to be very potent carcinogens and are known to be produced during some combustion processes (see, for example, CARB 1984). There are, however, very few data on PCDD/PCDF concentration in salt and ash that have been collected at incinerators similar in design and operation to the OWMC facility, i.e., HIM Biebesheim, Kommunekemi, and SAKAB. Given this limitation, the following equation was used to estimate

PCDD/PCDF concentrations in salt and ash generated by the OWMC incinerator:

$$C_{Dp} = \frac{C_{Ds}}{C_{Ps}}$$

Where:

C_{Dp} = concentration of PCDD/PCDF in particulate matter, mg/mg

C_{Ds} = concentration of PCDD/PCDF in the stack emissions (expressed as 2,3,7,8-TCDD equivalents, see Appendix H), mg/m^3

C_{Ps} = concentration of particulate matter in the stack emissions, 15 mg/m^3 (Monenco 1987)

The value for C_{Ds} cab be calculated using the following equation:

$$C_{Ds} = \frac{3\ 600\ (Q_{Ds})(t_T)}{(F)(T_O)}$$

Where:

C_{Ds} = concentration of PCDD/PCDF in stack emissions (expressed as 2,3,7,8-TCDD equivalents), g/Nm^3

Q_{Ds} = projected emission rate for PCDD/PCDF (expressed as 2,3,7,8-TCDD equivalents), g/s

t_T = hours in a year, hr/a

t_o = period of incinerator operation, hr/a

F = flue gas emission rate, Nm^3/hr

The following values were used in the risk assessment:

Q_{Ds} = 3.1×10^{-9} g/s (see Table H-3, Appendix H)

t_T = 8 760 hr/a

t_o = 7 000 hr/a (Monenco 1987)

$F = 22\ 860\ \text{Nm}^3/\text{a}$ (Monenco 1987)

Therefore,

$$C_{Ds} = \frac{(3\ 600)(3.1 \times 10^{-9})(8\ 760)}{(7\ 000)(22\ 860)}$$

$$C_{Ds} = 6.1 \times 10^{-10}\ \text{g/Nm}^3,\ \text{or}\ 6.1 \times 10^{-7}\ \text{mg/Nm}^3$$

Based on this value, the concentration of PCDD/PCDF in particulate matter is:

$$C_{Dp} = \frac{6.1 \times 10^{-7}}{15}$$

$$C_{Dp} = 4.1 \times 10^{-8}\ (\text{mg PCDD/PCDF})/(\text{salt and ash})$$

Therefore, the salt and flyash is 0.041 parts per million, expressed on a 2,3,7,8-TCDD equivalent basis. By using this approach, it was implicitly assumed that the particulate matter removed in the air pollution control system would have the same PCDD/PCDF concentration as the particulates emitted from the incinerator stack.

C.3.1.3 Residues Generated by the Solidification Plant

Residues produced by the OWMC solidification plant will represent the largest quantity of material deposited in the engineered landfill. The solidification plant will mix solid inorganic wastes received from off-site generators, solid residues generated by the physical/chemical treatment plant, and various reagents to produce a physically stable matrix designed to limit infiltration and leachate generation.

In order to characterize the composition of the residues produced by the solidification plant, it was first necessary to determine the identity and concentration of chemicals in both the wastes to be received from off-site generators and in the solid residues produced by the physical/chemical plant. The methodology developed for estimating contaminant concentrations for these two sources of waste are outlined below.

Chemical Loadings from Directly Solidified Off-Site Wastes. The estimation of contaminant concentrations in the wastes to be sent directly to solidification without treatment in the physical/chemical plant is discussed in Appendix B. The data used to estimate contaminant concentrations in the wastes were derived from studies conducted by CANVIRO Consultants (1985) and Franklin Associates. These contaminant concentrations were multiplied by the total quantity of waste to develop estimates of chemical loadings to the solidification unit from off-site generators. The quantities of surrogate chemicals in these wastes are shown in Table C-25.

Chemical Loadings from Wastes Initially Treated in the Physical/Chemical Plant. To estimate the quantities of specific chemicals in the semi-solid residues sent from the

TABLE C-25

SURROGATE CHEMICALS IN THE INORGANIC WASTES SENT DIRECTLY TO THE SOLIDIFICATION PLANT

Chemicals	Quantity (t/a)
Metals	
Arsenic	0.08
Barium	0.05
Cadmium	4.1
Chromium	304.4
Copper	912.9
Lead	923.4
Mercury	2.6
Nickel	229.0
Silver	0.98
Zinc	1 765.4
Inorganics	
Cyanide	1.0
Fluoride	0.01
Nitrate	4.9
Organics	
Benzo(a)anthracene	0.006
Benzo(a)pyrene	0.003
Benzene	0.04
Cresols	0.15
Dichlorodifluoromethane	0.1
Ethanol	0.1
Formaldehyde*	3.1
Naphthalene	0.12
PCBs	0.007
Petroleum distillates	3.9
Phenols*	5.54
Tetrachloroethylene	0.25
Toluene	58.3
1,1,1-Trichloroethane	0.53
Trichloroethylene	0.12
Trichlorotrifluorethane	0.19
Xylene	0.75

---

* According to the engineering consultants to OWMC, approximately 95 percent of the phenol and formaldehyde reported in the CANVIRO data base is in the form of a phenol-formaldehyde resin (Monenco 1987a). Since such a resin would have much different leaching characteristics than phenol or formaldehyde, the engineering consultants have estimated loadings to represent only the portion of these chemicals which may be liquid.

physical/chemical treatment plant to the solidification unit, a more complex procedure was required. This procedure involved:

- Prediction of the specific chemicals in the inorganic wastes to be received by the physical/chemical plant; and
- An analysis of the performance of the various physical/chemical processes in treating the streams containing these chemicals.

In the analysis, it was conservatively assumed that the entire quantity of each inorganic contaminant, other than cyanide, would ultimately reside in the solid residues sent to the solidification plant. These quantities were calculated in Appendix B. It has been determined by the engineering consultants that the chemical oxidation unit will destroy nearly all of the cyanide in the wastes received by the physical/chemical treatment plant, and as a result, of the 22.9 t/a of cyanide received by the plant, only approximately 0.5 kg/a will be present in the residues sent to solidification (Monenco 1987a).

Furthermore, over 400 t/a of nitrates would be predicted to be received by the physical/chemical treatment plant, based on information in the CANVIRO data base. Since this material is a valuable commodity, the engineering consultants to OWMC have determined that it is likely that little nitrate would actually be treated in the physical/chemical plant.

For the organic contaminants in the waste feed to the physical/chemical treatment facility, the extent to which an organic will partition to the solid residues is dependent, in part, upon its physical and chemical properties. In order to predict the concentrations of specific organics in the solid residues generated by the physical/chemical plant, the treatment performance for organic contaminant separation was assessed on an individual stream basis (Monenco 1987a).

The streams reported by the CANVIRO data base to contain specific organic chemicals are listed in Table C-26. The quantity of each of the streams was prorated to account for potentially hazardous organic material that was not defined for the purposes of the risk assessment. This prorating factor was calculated to be 5.2, as shown in Section C.2.2.1.

The performance assessment conducted by the engineering consultants was based on treatability data compiled through a review of the scientific literature (see Monenco 1986c, 1987) or developed in studies in studies commissioned by OWMC (Zenon 1987). These tests were undertaken to evaluate the level of treatment provided by the various physical/chemical plant process units. Using the results of these tests, the contaminant concentrations in both the liquid and solid phases of the waste were estimated for each physical/chemical operation (Monenco 1987a).

TABLE C-26

CONTAMINANT CONCENTRATIONS IN SPECIFIC INORGANIC STREAMS RECEIVED BY THE PHYSICAL/CHEMICAL TREATMENT PLANT

Stream	Contaminant	Concentration (mg/l)	Prorated Stream Quantity (t/a)
I-1	Phenol	1 000	7371.0
I-2	Toluene	10 000	5353.1
I-3	Phenol	5 000	5353.1
I-4	Styrene	5 000	8233.9
I-5	Acrylonitrile	5 000	8233.9
I-6	Phenol	1 000	137.4
I-7	Toluene	40 000	5024.9
I-8	Phenol	5 000	5024.9
I-9	Toluene	10 000	1741.7
I-10	Phenol	5 000	1741.7
I-11	Toluene	10 000	5226.8
I-12	Phenol	5 000	5226.8
I-13	Toluene	10 000	1741.7
I-14	Phenol	5 000	1741.7
I-15	Toluene	10 000	260.1
I-16	Phenol	10 000	260.1

According to the facility design, the semi-solid filter cake produced in the coagulation/precipitation unit of the physical/chemical treatment plant will be sent to solidification. Both the solid and liquid portions of the filter cake could contain trace levels of organic chemicals initially present in the inorganic wastes. Therefore, the total quantity of a chemical in the filter cake was calculated based on the following formula:

$$M_{i,T} = M_{i,S} + M_{i,L}$$

Where:

$M_{i,T}$ = mass of chemical i in the filter cake, t

$M_{i,S}$ = mass of chemical i in the solid portion of the filter cake, t

$M_{i,L}$ = mass of chemical i in the liquid portion of the filter cake, t

This equation can be express on an annual basis as follows:

$$Q_{i,T} = \frac{R}{1\ 000} [f_S\ C_{i,S} + (1-f_S)\ C_{i,L}]$$

Where:

$Q_{i,T}$ = quantity of chemical i in the filter cake, kg/a

R = total quantity of filter cake, t/a

f_S = fraction of the filter cake that is solid, dimensionless

$C_{i,L}$ = contaminant concentration in the liquid fraction, mg/l

$C_{i,S}$ = contaminant concentration in the solid fraction, µg/g

The following assumption was made:

f_S = 0.3 (Monenco 1987a)

It was further assumed that 10 percent of each inorganic stream would be composed of solid material (Monenco 1987a). Therefore, the total quantity of filter cake, R, was calculated for each stream examined using the following equation:

$$R = \frac{(0.1)(S)}{f_S}$$

Where:

S = total flow of stream (t/a)

The contaminant quantities in filter cake for each stream reported to contain organics were calculated using this approach. The calculations are summarized in Table C-27.

The only organic chemicals that were not examined on an individual stream basis were those identified in the Franklin data base as possible constituents of waste oil. Since "oil and grease" is expected in a relatively large number of inorganic streams to be received at the physical/chemical plant, it was not feasible to conduct a stream-by-stream evaluation of the partitioning of organic chemicals in oil to the solid residues. The quantities of oil contaminants likely to reside in the solid residues sent from the physical/chemical treatment plant to the solidification plant were therefore estimated by the engineering consultants to OWMC (Monenco 1987a). These quantities are listed in Table C-28.

TABLE C-27

CONTAMINANT QUANTITIES IN RESIDUES TO SOLIDIFICATION[1]

Stream	Contaminant	Contaminant Concentration in Liquid $C_{i,L}$ (mg/l)	Contaminant Concentration in Solid $C_{i,S}$ (ug/g)	Residue to Solidification R, (t/a)	Contaminant Quantity in Residue to Solidification Q_T (kg/a)
I-1	Phenol	5	30	2 457	31
I-2	Toluene	2	20	1 768	13
I-3	Phenol	5	30	1 784	22
I-4	Styrene	2	70	2 732	61
I-5	Acrylonitrile	ND	ND	---	---
I-6	Phenol	5	30	46	0.6
I-7	Toluene	2	20	1 609	12
I-8	Phenol	5	30	1 675	21
I-9	Toluene	2	20	575	4.3
I-10	Phenol	5	30	581	7.3
I-11	Toluene	2	20	1 726	13
I-12	Phenol	5	30	1 742	22
I-13	Toluene	2	20	575	4.3
I-14	Phenol	5	30	581	7.3
I-15	Toluene	2	20	86	0.6
I-16	Phenol	5	30	87	1.1

---

[1] Derived from Monenco 1987a.

ND: Not detected.

TABLE C-28

"OIL AND GREASE" CONSTITUENTS SENT TO SOLIDIFICATION FROM THE PHYSICAL/CHEMICAL TREATMENT PLANT

Contaminant	Concentration in "Oil and Grease"[1] (ppm)	Quantity to Solidification from Physical/Chemical[2] (kg/a)
Arsenic	18	1.1
Barium	251	15
Cadmium	10	0.6
Chromium	35	2.1
Lead	1 200	71
Zinc	1 130	66
Dichlorodifluoromethane	640	160
Trichlorotrifluoroethane	1 300	330
1,1,1-Trichloroethane[3]	3 500	170
Trichloroethylene	800	200
Tetrachloroethylene	1 600	410
Benzene	300	76
Toluene[3]	4 500	120
Xylene[3]	3 200	160
Benzo(a)anthracene	40	10
Benzo(a)pyrene	16	4
Naphthalene	800	200
PCBs	50	13

---

[1] Based on Franklin Associates (1985). See Appendix B.

[2] For organics, based on 255 t/a "oil and grease" to physical/chemical treatment (49 t/a multiplied by a prorating factor of 5.2). For metals, based on a 59 t/a "oil and grease" (49 t/a multiplied by a prorating factor of 1.2). See Appendix B for details.

[3] Contaminant quantities calculated by the engineering consultants based on treatability test data (Monenco 1987a).

C.3.1.4 Total Loadings to the Engineered Landfill

The total chemical quantities sent to the proposed OWMC engineering landfill were calculated by combining the estimates developed for incinerator slag, incinerator salt and ash, and residues produced by the solidification plant. These quantities are presented in Table C-29. Corresponding mass fractions in the wastes sent to solidification are presented in Table C-30.

C.3.2 ESTIMATION OF CHEMICAL CONCENTRATIONS IN LEACHATE

The infiltration of precipitation into the OWMC engineered landfill will result in the partial solubilization of contaminants to produce an aqueous liquid known as leachate. As part of the risk assessment, an approach was developed to estimate the concentrations of chemicals in leachate based on the predicted concentrations in the landfilled material (WTC 1987). The approach developed in the assessment was based on the following assumptions:

- The solidified material is a porous solid.
- The composition of the pore solution within the solidified material is equivalent to the composition of the leachate generated in the landfill.
- The pore solution is in continuous chemical equilibrium with the solid portion of the solidified material.

TABLE C-29

SURROGATE CHEMICALS IN THE RESIDUES SENT TO THE SOLIDIFICATION PLANT

Chemicals	Loadings (t/a) Slag	 Salt and Ash	 Direct Solidification	 Physical/ Chemical Residues
Metals				
Antimony	--	--	--	6.7
Arsenic	--	--	0.08	6.8
Barium	278	2.5	0.05	0.02
Cadmium	--	2.2	4.1	15.5
Chromium	289	1.3	304.4	193.5
Copper	220	7.0	912.9	153.7
Lead	14.6	42.1	923.4	12.5
Mercury	--	0.1	2.6	0.77
Nickel	14.6	1.1	229.0	145.3
Silver	0.1	--	1.0	29.6
Zinc	7.3	77.6	1 765.4	753.4
Inorganics				
Cyanide	--	--	*	0.0005
Fluoride	--	--	0.01	0.07
Nitrate	--	.26	4.9	1.6
Organics				
Benzo(a)anthracene	--	--	0.006	0.010
Benzo(a)pyrene	--	--	0.003	0.004
Benzene	--	--	0.04	0.076
Cresols	--	--	.15	--
Dichlorodifluoromethane	--	--	0.1	0.16
Ethanol	--	--	0.1	--
Formaldehyde	--	--	3.1	--
Naphthalene	--	--	0.12	0.20
PCBs	--	--	0.007	0.013
Petroleum Distillates	--	--	3.9	--
Phenols	--	--	5.54	0.11
Styrene	--	--	--	0.061
Tetrachloroethylene	--	--	0.25	0.41
Toluene	--	--	58.3	0.17
1,1,1-Trichloroethane	--	--	0.53	0.17
Trichloroethylene	--	--	0.12	0.20
Trichlorotrifluorethane	--	--	0.19	0.33
Xylene	--	--	0.75	0.16
PCDD/PCDF	--	0.00027	--	--

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* According to the CANVIRO data base, a contaminant identified as cyanate may go directly to solidification. If cyanide is received, it would have to be sent to the physical/chemical treatment plant.

TABLE C-30

SURROGATE CHEMICALS IN THE SOLIDIFIED MATERIAL SENT TO THE ENGINEERED LANDFILL

Chemicals	Quantity (t/a)	Mass Fraction in Solidified Material*
Metals		
Antimony	6.7	5.9 E-05
Arsenic	6.9	6.1 E-05
Barium	280.6	2.5 E-03
Cadmium	21.8	1.9 E-04
Chromium	788.2	7.0 E-03
Copper	1 293.6	1.1 E-02
Lead	993.0	8.8 E-03
Mercury	3.5	3.1 E-05
Nickel	390.1	3.5 E-03
Silver	30.7	2.7 E-04
Zinc	2 603.7	2.3 E-02
Inorganics		
Cyanide	0.0005	4.4 E-09
Fluoride	0.1	7.1 E-07
Nitrate	6.8	6.0 E-05
Organics		
Benzo(a)anthracene	0.02	1.4 E-07
Benzo(a)pyrene	0.01	6.3 E-08
Benzene	0.12	1.0 E-06
Cresols	0.15	1.3 E-06
Dichlorodifluoromethane	0.26	2.3 E-06
Ethanol	0.10	8.8 E-07
Formaldehyde	3.1	2.8 E-05
Naphthalene	0.32	2.8 E-06
PCBs	0.02	1.8 E-07
PCDD/PCDF (2,3,7,8-TEQ)	0.00027	2.4 E-09
Petroleum distillates	3.90	3.5 E-05
Phenols	5.65	5.0 E-05
Styrene	0.06	5.4 E-07
Tetrachloroethylene	0.66	5.8 E-06
Toluene	58.5	5.2 E-04
1,1,1-Trichloroethane	0.69	6.2 E-06
Trichloroethylene	0.32	2.8 E-06
Trichlorotrifluorethane	0.52	4.6 E-06
Xylene	0.91	8.1 E-06

---

* Based on 113 000 t/a solidified material.

Since the leachate composition will depend upon the physical and chemical characteristics of the contaminants present in the solidified material, chemicals were categorized into three groups:

- Class A. Chemicals which are completely soluble in the pore solution of the solidified material.
- Class B. Chemicals for which the concentration in the pore solution is controlled by a linear adsorption isotherm; i.e., the concentration in the solidified material divided by the concentration in the pore solution is equal to a constant greater than zero. This constant is referred to as the adsorption coefficient (Kd).
- Class C. Chemicals for which the concentration in the pore solution is controlled by the solubility of a slightly soluble salt.

The classification of the chemicals examined in the risk assessment is shown in Table C-31.

For chemicals in Classes A and B, the following relationship served as the basis for estimating concentrations in leachate:

$$C_L = \frac{C_s}{K_d}$$

Where:

C_L = chemical concentration in leachate, mg/l

C_s = chemical concentration in solid material, (mg/kg)

K_d = distribution coefficient, (mg/kg)/(mg/l)

TABLE C-31

CHEMICAL CLASSIFICATION FOR THE LANDFILL ANALYSIS

Chemicals	Chemical Category
Metals	
Antimony	C
Arsenic	C
Barium	C
Cadmium	C
Chromium	C
Copper	C
Lead	C
Mercury	C
Nickel	C
Zinc	C
Inorganics	
Cyanide	B
Fluoride	A
Nitrate	A
Organics	
Acrylonitrile	A
Benzo(a)anthracene	B
Benzo(a)pyrene	B
Benzene	B
Cresols	B
Dichlorodifluoromethane	A
Ethanol	A
Formaldehyde	A
Naphthalene	B
PCBs	B
PCDD/PCDF (2,3,7,8-TEQ)	B
Petroleum distillates	B
Phenols	B
Styrene	B
Tetrachloroethylene	B
Toluene	B
1,1,1-Trichloroethane	B
Trichloroethylene	B
Trichlorotrifluorethane	A
Xylene	B

Using the above relationship, an equation for relating the chemical concentration in leachate to the chemical concentration in the solidified material was developed:

$$C_{i,L} = \frac{(1 \times 10^6)(M_i)}{W + (1-W)(K_d)}$$

Where:

$C_{i,L}$ = chemical concentration in leachate, mg/l

M_i = mass fraction of chemical in the solidified material, dimensionless

W = water content in solidified material, dimensionless

K_d = distribution coefficient (mg/kg)/(mg/l)

The following assumption was made in the analysis:

W = 0.40 (Wastewater Technology Centre, 1986)

It was assumed that chemicals in Class A would be completely soluble in the pore solution of the solidified waste, i.e., K_d would approach 0. K_d values for the chemicals in Class B were estimated by the Wastewater Technology Centre (WTC) and are presented in Table C-32.

For contaminants in Class C, the pore solution concentration is independent of the concentration in the solidified waste. For these contaminants, the concentration in the leachate was determined by the chemical speciation, e.g., carbonate, hydroxide, sulphide (Monenco 1987a). Equilibrium leaching tests were conducted for certain Class C chemicals

TABLE C-32

ASSUMED ADSORPTION COEFFICIENT (K_d) VALUES

Chemicals	Adsorption Coefficient(K_d) (mg/kg)/(mg/l)
Cyanide	20
Benzo(a)anthracene	1 500
Benzo(a)pyrene	1 500
Benzene	100
Cresols	500
Naphthalene	1 500
PCBs	5 000
PCDD/PCDF (2,3,7,8-TEQ)	500 000
Petroleum distillates	500
Phenols	100
Styrene	100
Tetrachloroethylene	100
Toluene	100
1,1,1-Trichloroethane	50
Trichloroethylene	100
Xylene	1 000

Source: Derived by OWMC from data provided by Wastewater Technology Centre, 1987.

considered in the landfill analysis to estimate pore solution concentrations for these contaminants. These results are summarized in Table C-33.

Based on the approaches described above, leachate concentrations were estimated for each chemical examined in the risk assessment. These concentrations are presented in Table C-34.

C.4 RELEASES ASSOCIATED WITH ON-SITE UPSETS AND ACCIDENTS

Contaminant emissions resulting from on-site upsets and accidents were previously estimated in the Phase 4A generic risk assessment (ENVIRON 1985). These emission estimates have not been revised in the current risk assessment. However, the current Phase 4B risk assessment has been expanded to include an estimate of the frequency with which an on-site tank farm fire could occur. Such estimates were not developed for the other accident and upset scenarios examined since the results of Phase 4A generic risk assessment indicated that emissions associated with these events would not pose a significant risk to public health (ENVIRON 1985).

TABLE C-33

PORE SOLUTION CONCENTRATIONS FOR SLIGHTLY SOLUBLE SALTS

Contaminant	Concentration (mg/l)
Antimony	5×10^{-2}
Arsenic	1×10^{-2}
Barium	3×10^{-1}
Cadmium*	1×10^{-2}
Chromium	1×10^{-2}
Copper	1×10^{-1}
Lead	1×10^{-2}
Mercury	1×10^{-4}
Nickel	1×10^{-1}
Zinc	1×10^{-1}

Source: Based on data provided by Wastewater Technology Centre (Monenco 1987a). Data for silver not available.

* Not detected in the pore solution in tests conducted by the Wastewater Technology Centre. Pore solution conservatively estimated using the detection limit.

TABLE C-34

SURROGATE CHEMICAL CONCENTRATIONS IN LEACHATE

Chemicals	Leachate Concentration (mg/l)
Metals	
Antimony	0.05
Arsenic	0.01
Barium	0.3
Cadmium	0.01
Chromium	0.01
Copper	0.1
Lead	0.01
Mercury	1.0 E-04
Nickel	0.1
Zinc	0.1
Inorganics	
Cyanide	3.6 E-04
Fluoride	1.8
Nitrate	150
Organics	
Benzo(a)anthracene	1.57 E-04
Benzo(a)pyrene	6.98 E-05
Benzene	0.017
Cresols	0.0044
Dichlorodifluoromethane	5.8
Ethanol	2.2
Formaldehyde*	69.0
Naphthalene	0.0032
PCBs	5.9 E-05
PCDD/PCDF (2,3,7,8-TEQ)	7.6 E-09
Petroleum distillates	0.12
Phenols	0.83
Styrene	0.009
Tetrachloroethylene	0.097
Toluene	8.6
1,1,1-Trichloroethane	0.20
Trichloroethylene	0.047
Trichlorotrifluorethane	11.5
Xylene	0.013

---

* Leachate concentration based on liquid formaldehyde. According to the engineering consultants, actual loadings may be in the form of a polymerized resin (Monenco 1987a). Therefore, leachate concentrations may be significantly overestimated.

C.4.1 TANK FARM FIRE

Two scenarios involving a tank farm fire resulting in vaporization and subsequent emission of the selected surrogate chemical, methylene chloride, to the atmosphere were examined. In the first scenario, a leak in one of three halogenated organic waste tanks results in a spill of waste which collects in the diked area immediately surrounding the tank. This waste burns and volatilizes the remainder of the waste in the tank. In the second scenario, material from one of six organic sludge tanks spills and ignites. Radiant heat transfer to the adjacent halogenated organic storage tanks results in vaporization of the tank contents. The two scenarios are described in greater detail in the following subsections.

C.4.1.1 Emissions

Fire in the Halogenated Organic Waste Storage Tank Area. The three halogenated organic waste storage tanks at the OWMC facility will be located within a 34 meter by 5.2 meter diked area (Monenco 1987). The tanks will be separated from each other by internal dikes 0.4 meters high and thus a spill would be confined to the area surrounding the leaking tank.

In this scenario, it has been envisioned that a 8 m^3 tank containing the selected surrogate chemical, methylene chloride, is exposed to a fire in the diked area surrounding the tank. The contents of the tank are boiled off and vented at high elevation away from the diked area. The emission rate was estimated by the method of the American Petroleum Institute (API, 1976) as:

$$Q_i = \frac{21\ 000\ (F)(A_w)^{0.82}}{\Delta_H}$$

Where:

Q_i = emission rate, lb/hr

F = insulation factor = 1.0 for a bare vessel

A_w = wetted surface area of tank, ft^2

Δ_H = heat of vaporization, 139 Btu/lb

The following assumptions were made (Monenco 1987):

- the wetted surface area of the tank is 176.7 ft^2
- the tank capacity is 8 m^3

Therefore,

$$Q_i = \frac{21\ 000\ (1)(176.7\ ft^2)^{0.82}}{(139\ Btu/lb)}$$

$$Q_i = 1.05 \times 10^4\ lb/hr$$

$$Q_i = 1\ 328\ g/s$$

This emission rate has been calculated assuming that the halogenated waste storage tanks will be uninsulated.

Fire in Adjacent Organic Sludge Waste Area. Sludge wastes at the OWMC facility will be contained in six tanks within a 34 meter by 15.35 meter diked area. The dike separating the halogenated organic tanks from the organic sludge tanks will be 0.4 meters high. Individual tanks within the organic sludge tank area will not be separated by dikes.

In this scenario, it has been envisioned that one completely filled tank (120 cubic meters) of organic sludge waste leaks. The resulting spilled material distributes evenly throughout the entire diked area surrounding the sludge tanks, creating a pool with a depth of 0.23 meters, and then ignites. The results of a hazard analysis study performed for this scenario (Battelle 1987) suggests that the expected burning rate would be approximately 5 mm/min. Therefore, the total burning time for a spill of a single tank would be approximately 46 minutes (Battelle 1987).

If the burning sludge waste completely covers the diked area, the flame will be at least 15 meters high and 34 meters wide (Battelle 1987). Since these dimensions are very large in comparison to the dimensions of the halogenated organic waste tanks and compared to the distance between those tanks and the dike, the flame can be considered to be of infinite height and width.

Under these conditions, radiant heat transfer to the halogenated organic waste tank can be calculated as follows (Battelle 1987):

$H_t = A_e H_f$

Where:

H_t = total heat transfer rate to tank, KJ/hr

A_e = effective area of the tank, m^2

H_f = radiant heat flux, kJ/hr m^2

Based on the hazard analysis study, the following assumptions were made (Battelle 1987):

- The effective area of the tank is one-half the cylindrical surface, 2.9 meters high by 2.1 meters in diameter, multiplied by one-half for the semi-infinite flame, and multiplied by 0.8 for the assumed emissivity of the tank surface:

  $A_e = (2.1\ m)(\pi)(2.9\ m)(0.5)(0.5)(0.8)$

  $A_e = 3.83\ m^2$

- The organic sludge burns with a sooty flame, with a flame surface temperature of 870°C. The resultant radiant heat flux, H_f, will be 354 000 kJ/hr.

  Therefore:

  $H_t = (3.83\ m^2)(354\ 000\ kJ/hr)$

  $H_t = 1\ 354\ 000\ kJ/hr$

Before the selected surrogate chemical, methylene chloride, begins to vaporize, it must be heated from the ambient temperature in the storage tank to its boiling point at 10 psig (Monenco 1987). The time required to heat the contents of the tank to boiling is given by:

$$t_b = \frac{V_w\ \rho\ \Delta H_s\ (T_b - T_a)}{H_t}$$

Where:

t_b = time required to reach boiling point at 10 psig, hr

V_w = volume of waste in tank, m^3

ρ = density of waste, kg/m^3

ΔH_s = specific heat of waste, kJ/kg

T_b = boiling temperature at 10 psig, °C

T_a = ambient temperature in tank

H_T = heat transfer rate to tank, kJ/hr

Based on the facility design (Monenco 1987) and the physical/chemical properties of methylene chloride (Battelle 1987), the following assumptions were made:

V_w = 8 m^3

ρ = 1 134 kg/m^3

ΔH_s = 1.206 kJ/kg

T_b = 53°C

T_a = 21°C

Thus the time interval from initiation of a fire in the organic sludge waste area to the boiling of methylene chloride in the halogenated organic storage tanks becomes:

$$t_b = \frac{(8\ m^3)(1\ 134\ kg/m^3)(1.206\ kJ/kg)(53°C - 21°C)}{1\ 354\ 000\ kJ/hr}$$

t_b = 0.25 hr, or 15 min.

As previously indicated, the total burning time for a spill of a single organic sludge tank would be 46 minutes. Therefore, during the 31 minutes following heating of the methylene chloride to its boiling temperature at 10 psig, the heat transfer to the tank will result in the vaporization of methylene chloride. During this time interval, the methylene chloride emission rate will be:

$$Q_i = \frac{H_t}{\Delta H_L}$$

Where:

Q_i = emission rate, g/s

H_T = heat transfer rate to tank; 1 345 000 kJ/hr

ΔH_L = latent heat of vaporization of methylene chloride, 314 kJ/kg (Battelle 1987)

Therefore, the emission rate will be:

$$Q_i = \frac{(1\ 345\ 000 \text{ kJ/hr})(1 \text{ hr}/3\ 600 \text{ sec})}{(314 \text{ kJ/kg})(\text{kg}/1\ 000 \text{ g})}$$

$$Q_i = 1\ 190 \text{ g/s}$$

This emission rate is slightly lower than that predicted under conditions of the first scenario and would occur for a shorter period of time, approximately 31 minutes.

C.4.1.2 Frequency

In the Phase 4A generic risk assessment (ENVIRON 1985), it was determined that the level of human exposure to methylene chloride released during tank farm fire could be significant, although not likely to cause incapacitating illness or death. Given these results, it was recognized that an estimate of the frequency of a tank farm fire would be necessary to develop a more complete understanding of the hazards posed by such an event. Therefore, a fault tree analysis was performed in Phase 4B to determine how often a tank farm fire might be expected to occur at the proposed OWMC facility (Battelle 1987). The results of this study are summarized below.

A tank farm fire causing (at least partial) boil-off of the contents of one of the halogenated organic waste tanks was modeled as resulting from a sequence of two events:

- A leak or spill from a halogenated organic waste tank or an adjacent organic sludge waste tank; and
- Ignition of the spilled material.

For any individual tank, the frequency of a fire can be expressed mathematically as:

$$f_T = f_L f_i$$

Where:

f_T = frequency of a fire resulting from a leak in any given tank; a^{-1}

f_L = frequency of a spill of leak from the tank; spills/a

f_i = frequency that a spill will be ignited; spill^{-1}

Based on an event tree/fault tree analysis, the frequency of a leak or spill from equipment and/or human error was estimated to be 2.6 x 10^{-2} spills/a, i.e., f_L = 2.6 x 10^{-2} spills/year (Battelle 1987). The frequency of ignition, f_i, was estimated to be approximately one in every 25 spills, based on data from oil refinery operations (Battelle 1987).

Therefore:

$$f_T = (2.6 \times 10^{-2} \text{ spills/a})(1/25 \text{ spill}^{-1})$$

$$f_T = 1.04 \times 10^{-3} \text{ a}^{-1}$$

Thus a fire resulting from leakage of any individual tank would be expected to occur about once in 960 years. If it is assumed that all six organic sludge waste tanks and all three halogenated organic waste tanks are in full usage, then nine tanks could potentially contribute to the probability of a fire. Therefore, the frequency of a fire with volatilization of halogenated organic waste (modeled here as methylene chloride) was estimated not to exceed about once in 100 years.

It should be noted that the analysis conducted did not account for any mitigative measures to reduce the hazard of ignition, even after the spill takes place. Such measures would include (Battelle 1987):

- Detection of the spill before ignition;
- Prevention of immediate/delayed ignition;
- Fire suppression before vapor boil-off is significant;

- Using fireproof tank insulation; and
- Segregating groups of halogenated and sludge tanks.

In light of these considerations, the calculated frequency for a tank farm fire is conservative, i.e., the scenario evaluated would be expected to occur less frequently than once in 100 years.

C.4.2 MIXING OF INCOMPATIBLE WASTES

The generation of hydrogen sulfide as a result of mixing of incompatible wastes was examined in earlier studies conducted for OWMC on the selection of a buffer zone for the proposed facility (M.M. Dillon 1984). In this scenario it was envisioned that 4 percent sulfuric acid would be accidentally mixed with sulfide contaminated material in the physical/chemical treatment building. Hydrogen sulfide gas would consequently be emitted first into the building and then to the atmosphere through a ventilation system.

This event has been simulated in the laboratory on a reduced scale by mixing 4 percent sulfuric acid with zinc sulfide. Based on the results of the experiment, the emission rate for this scenario was estimated to be 0.462 g/s over a 30 minute interval (M.M. Dillon 1984).

C.4.3 INCINERATOR SCRUBBER FAILURE

In the event of a flue gas scrubber failure, the emergency power will be designed to resume operations in less than 30 seconds (Monenco 1987). Gas flow would decrease to 10% of normal in 4 minutes following failure. In the calculation of emission rates it was assumed that at a facility capacity of 150 000 t/a, one incinerator would be in operation and at 300 000 t/a, two incinerators would be operational. Based on data supplied by Monenco, the emission rates for the two minutes of failure will be:

Q_i = (Average volumetric flow rate) (Acid gas concentrations)

= $(V)(C_g)$

Where:

Q_i = emission rate, g/s

V = average volumetric flowrate, 6.7 m^3/s

C_g = acid gas concentrations, g/m^3

The emission rates at a facility capacity of 150 000 t/a are presented in Table C-35. At 300 000 t/a, it was conservatively assumed that power failure would result in scrubber failure for both incinerator units.

C.4.4 CYANIDE TREATMENT - UNCONTROLLED pH

In the risk assessment, emissions from the treatment of cyanide-bearing wastes under abnormal conditions, i.e., when

TABLE C-35

EMISSION RATES DURING FLUE GAS SCRUBBER FAILURE

Contaminant	Emission Rate (g/s)
Hydrogen Chloride	84.0
Sulfur Dioxide	65.2
Hydrogen Fluoride	1.9
Nitrogen Oxides	1.9

the pH control systems fails, were examined. During these conditions, the concentration of hydrogen cyanide in equilibrium with the solution would increase. For example, a reduction from the normal pH of 9.5 to 7.5 would result in an initial higher HCN vapor concentration (593 ppm) in the gas (Monenco 1987). Vapor emission over a 6 hour period would be:

$$V = \frac{(0.5\ \text{m}^3/\text{min})\ (5.93 \times 10^{-4}\ )}{2}$$

$$= 1.48 \times 10^{-4}\ \text{m}^3/\text{min} \times 60\ \text{min/h} \times 6\ \text{h/batch}$$

$$= 5.34 \times 10^{-2}\ \text{m}^3/\text{batch}$$

Therefore, the emission rate would be:

$$Q_i = \frac{(760\ \text{mm Hg})(5.34 \times 10^{-2}\ \text{m}^3/\text{batch})(27.03\ \text{g/mol})(1-0.09)}{0.0624\ (\text{m}^3\ \text{mm Hg})/(\text{mol k})\ 273\text{K}}$$

$$= 6.4\ \text{g/batch} \times \text{batch}/6\ \text{h} \times 1\ \text{h}/3600\text{s}$$

$$= 3.0 \times 10^{-4}\ \text{g/s}$$

C.4.5 SULFIDE TREATMENT - UNCONTROLLED pH

The removal of heavy metals from waste solutions may be accomplished by the addition of a sodium sulfide solution to a batch of the waste. In the alkaline state, this results in the precipitation of heavy metals as sulfides, and emission of hydrogen sulfide is negligible.

If, during abnormal conditions, the pH is allowed to fall to the acidic state the concentration of gaseous hydrogen sulfide in equilibrium with the waste solution increases. To develop an understanding of the consequences of such a pH excursion during sulfide treatment, the engineering consultants have predicted the hydrogen sulfide emission rate associated with the following illustrative scenario (Monenco 1987):

- As the result of equipment malfunction or operator error, the pH in the reaction vessel drops from its normal level of 9.5 to 5.0.
- The effective Na_2S concentration in the vessel at the time of the pH excursion is 0.01 percent.

At a facility capacity of 150 000 t/a, the following equation can be used to estimate the hydrogen sulfide emission rate:

$$Q_i = \frac{(P_o)(V)}{(R)(T)} (M_{H_2S}) (1-\eta)$$

Where:

Q_i = emission rate, g/s

P_o = standard condition pressure (760 mm Hg)

R = gas constant = 0.0624 (m^3.mm Hg)/(mol.K)

T = standard condition temperature (273 K)

V = volumetric flowrate of gas at standard condition

η = scrubber/adsorber efficiency

M_{H2S} = molecular weight, 34.08 g/mol

Based on facility design and operations (Monenco 1987), the following assumptions were made for calculation of emissions under abnormal pH situation:

- the adsorber efficiency is 90% ($\eta=0.9$)
- the volumetric flowrate of H_2S gas is 0.303 m^3/hr
- pH falls to 5

$$V = (0.303\ m^3/h)(h/3600\ s)$$

$$= 8.41 \times 10^{-5}\ m^3/s$$

Therefore, at 150 000 t/a plant capacity:

$$Q_i = \frac{(760\ mm\ Hg)(8.41 \times 10^{-5}\ m^3/s)(34.08\ g)(1-0.9)}{0.0624\ (m^3\ mm\ Hg)/(mol\ K)\ (273\ K)}$$

$$= 1.28 \times 10^{-2}\ g/s$$

C.4.6 SUMMARY OF EMISSION ESTIMATES

Emission estimates for all on-site accidents and upsets considered in this section are summarized in Table C-36.

C.5 TRANSPORTATION ACCIDENTS AND MALFUNCTIONS

Transportation accidents involving vehicles carrying special wastes, additives, or reagents to the proposed OWMC facility may occur along both local access roads and Provincial highways. In the Phase 4A generic risk assessment, it was concluded that accidents along the local access route were of greater concern due to the proximity of residences and because the relative concentration of OWMC-related traffic on these roads would be higher than along the Provincial highway

TABLE C-36

EMISSION ESTIMATES FOR ON-SITE UPSET SCENARIOS

Operations	Emission Rate @ 150 000 t/a (g/s)
Tank Farm Fire	
Methylene Chloride	1 328
Cyanide Treatment	
Hydrogen Cyanide	3.0 E-04
Sulfide Treatment	
Hydrogen Sulfide	1.3 E-02
Mixing of Incompatible Wastes	
Hydrogen Sulfide	0.46
Incinerator Scrubber Failure	
Hydrogen Chloride	84.0
Sulfur Dioxide	65.2
Hydrogen Fluoride	1.9
Nitrogen Oxides	1.9

system. Furthermore, it was determined in Phase 4A that the risks associated with spills of special wastes would be significantly greater than those posed by spills of additives and reagents (ENVIRON 1985). For these reasons, the risk assessment for the OWMC facility has been limited to an analysis of spills of special waste along the local access route.

In Phase 4A, a spill onto a roadway with subsequent volatilization of chemicals was evaluated. The risk assessment has been expanded in Phase 4B to include an examination of a spill of special waste into a stream. These two scenarios are discussed in this section.

C.5.1 SPILL ONTO A ROADWAY

The transportation scenario considered in Phase 4A assumed that a 18 tonne tank truck carrying a load of liquid waste is involved in a collision on the local access road, resulting in a tank rupture and release of the entire contents of the truck. The released liquid is assumed to spread across the road surface to form a pool from which the selected surrogate chemical, methylene chloride, is released to the atmosphere. The pool was assumed to be circular with the center of the circle coincident with the centerline of the road, and the diameter constrained by the width of the paved surface of the road (7.0 m). Clearly, different conditions (e.g., nature of the tank rupture, configuration of the road surface, shoulder or roadside ditches, will affect the size and shape of the pool.

C.5.1.1 Emission Estimate

The emission rate for chemicals released to the atmosphere from a pool of liquid on the roadway was calculated based on an equation developed by Stiver and MacKay (1982):

$$Q_i = \frac{(K_{g_i})(A)(P_i)(x_i)(M_i)}{(R)(T)}$$

Where:

Q_i = emission rate for species i, g/s

Kg_i = the gas mass transfer coefficient for species i, m/s

A = area of the spill, m^2

P_i = vapor pressure for species i at 25°C, mm-Hg

x_i = mole fraction for species i, mol/mol

M_i = molecular weight of species i, g/mol

R = gas constant, 0.0624 mm-Hg m^3/mol K

T = temperature, K

This equation is the same as that used to estimate fugitive releases resulting from on-site spills, and is discussed in detail in Section C.2.3.2.

The following assumptions were made in the analysis:

A = 38.5 m^2, based on a circular pool with the center of the circle coincident with the centerline of the road, and the diameter constrained by the width of the paved surface (7 m)

T = 298°K

P_i = 433 mm-Hg

X_i = 0.79, based on a mass fraction of 0.69 (Section B.5.2) and the relationship defined in Section C.2.3.

M_i = 84.9

K_g = 2.7×10^{-3}, (calculated using the equation of MacKay and Matsugu (1973) described in Section C.2.3.2.)

Based on the above assumptions, an emission rate of 162 g/s was calculated.

C.5.1.2 Frequency

The frequency of a truck accident-related spill of special wastes along the local access route has been calculated by the transportation consultants to OWMC (M.M. Dillon 1986). For calculation purposes, the local access route has been divided into a sequence of eight links, for which accident involvement data have been compiled individually. The total truck accident-related spill frequency for all links of the access route is given as (M.M. Dillon 1985):

$$S_f = \sum[(L_1)(A_{f1})(N)(f_{tg})(f_{sa})]$$

Where:

S_f = truck accident-related spill frequency, yr^{-1}

L_1 = length of link 1, km

A_{f1} = general vehicle accident frequency, accidents per vehicle per km

N = number of one-way truck deliveries to the OWMC facility per year

f_{tg} = ratio of accident frequency for trucks to the general vehicle accident frequency

f_{sa} = number of spills per truck accident.

Data for L_1 and A_{f1} have been compiled by M.M. Dillon (1986), and are summarized in Table C-37. Other assumptions have been made as follows:

- The annual number of special waste deliveries by truck will be 6 550 (Monenco 1987)
- f_{tg}, the accident frequency for trucks, is 1.37 times greater than the general vehicle accident frequency (M.M. Dillon 1986)
- f_{sa}, the number of spills per truck accident, is approximately 0.196 (Pilorusso 1983).

Based on these assumptions, the spill frequency associated with truck accidents was estimated to be 0.095 yr^{-1} for a facility capacity of 150 000 t/a, or about once in 10.5 years. This estimate, however, applies to a spill of any special waste being transported to the OWMC facility. Approximately 25 percent of the waste to be transported will be non-pumpable (Dillon 1985); therefore, the frequency of a spill of pumpable

TABLE C-37

TRANSPORTATION ACCIDENT DATA FOR THE OWMC FACILITY LOCAL ACCESS ROUTE

Link Number	Link Description	Length, L_l (km)	General Vehicle Accident Frequency, A_{fl} (Accidents per vehicle per km) x 10^6
1	QEW to RR 81	3.8	2.28
2	RR 81 to Moyer Road	0.7	5.43
3	Moyer Road to RR 73	1.1	0.71
4	RR 73 to Ninth Avenue	3.5	2.77
5	Ninth Avenue to RR 216	3.0	2.70
6	RR 216 to Highway 20	4.1	2.55
7	Highway 20 to Schram Road	5.1	1.33
8	Schram Road to OWMC	0.9	1.86

Source: M.M. Dillon 1986.

waste would be 0.75 x 0.095 yr^{-1}, or 0.071 yr^{-1}. This corresponds to once in about 14 years. Two more restrictive cases were also considered.

First, it has been estimated that only 8 percent of the special waste transported to the facility would be capable of causing adverse effects in the exposed off-site population (ENVIRON 1985). For these spills, the base frequency of 0.095 yr^{-1} for all special wastes must be reduced by a factor of 0.08, to approximately 0.0076 yr^{-1}. A spill with the potential to cause some type of effect in the exposed community is therefore predicted to occur approximately once in 132 years.

Next, it has also been estimated that only 0.8 percent of the special waste to be received by the OWMC facility would consist of methylene chloride, the most hazardous special waste (ENVIRON 1985). For transportation accident-related spills of methylene chloride, the base frequency of 0.095 yr^{-1} for all special wastes is therefore estimated to be reduced by a factor of 0.008, to approximately 0.00076 yr^{-1}. Spills of the most hazardous waste received at the OWMC facility would therefore be expected to occur approximately once in 1,320 years.

At the ultimate facility capacity of 300 000 t/a, the number of truck shipments of special wastes would double. Therefore, the predicted spill frequencies would double as well.

C.5.2 SPILL INTO SURFACE WATER

The local access route to the proposed OWMC facility crosses several streams, including Twenty Mile Creek, Ten Mile Creek, and Five Mile Creek. A spill which occurs near any of these crossings could result in a spill of hazardous waste into surface water. In the risk assessment, Twenty Mile Creek was selected for evaluation since it has a number of sensitive downstream uses, and is the largest stream along the preferred access route.

Bulk liquids, solids and sludges, and containerized waste will be transported to the proposed facility along various local and regional roads in trucks of varying types and sizes. In the Phase 4B risk assessment, a tank truck carrying a load of bulk liquid waste was assumed to be involved in a spill incident (accident or equipment failure) at the intersection of Regional Road 24 and Twenty Mile Creek. This spill incident results in a partial release of the contents of the tank truck directly into Twenty Mile Creek.

The spill scenario evaluated is likely to provide a conservative estimate of contaminant concentrations in the creek for the following reasons: 1) a spill directly into a river is considered to represent the most severe spill scenario for the surface water pathway; and 2) a spill of bulk liquids is considered to provide the least opportunity for spill containment and control relative to bulk sludges and solids, or containerized waste.

The hazard posed by chemicals spilled into surface waters depends, to a large extent, on the behavior of the chemicals in the water. Three categories of chemicals were considered in the Phase 4B risk assessment:

(1) "Mixers"	soluble compounds which rapidly mix with water;
(2) "Floaters"	insoluble (or slightly soluble) compounds which float on water; and
(3) "Sinkers"	insoluble (or slightly soluble) compounds which sink to the bottom on the creek.

The selection of surrogate chemicals used to represent each category of waste is discussed in Appendix B; the fate and transport of the compounds in Twenty Mile Creek is described in Appendix E. This section considers the potential magnitude of a spill and the estimated frequency of a release, into Twenty Mile Creek.

C.5.2.1 Magnitude of a Spill

Estimates of the magnitude of the spill were based on data taken from computer records maintained by the U.S. Department of Transportation (DOT) Office of Hazardous Materials Transportation. These records were accessed to determine the frequency of various size spills that met the following criteria:

- The spill occurred between January 1971 and October 1986;
- The spill was from a full size tanker (a minimum of 15,140 liters);
- The spill occurred during highway transit; and
- The spill involved liquid losses.

The distribution of releases from tankers under these conditions is presented in Table C-38. As indicated in the table, 14,475 incidents met the above criteria. The median quantity of material released was approximately 227 liters, 75 percent of the losses were less than approximately 1,514 liters; and 90 percent were less than approximately 11,355 liters. The largest spill involved was approximately 56,775 liters.

Three spill sizes were selected for evaluation in the current analysis: 230 liters (approximately 50 percent of the spills are greater than or equal to 230 liters), 1 500 liters (25% of the spills are greater than or equal to 1 500 liters). and 19 000 liters, (5 percent of the spills are greater than or equal to 19 000 liters).

TABLE C-38

DISTRIBUTION OF CARGO LOSSES FROM FULL SIZE TANKERS DURING HIGHWAY TRANSIT IN THE U.S. FROM 1971 TO OCTOBER 1986

Size of Spill (gallons)	Size of Spill (liters)	Number of Incidents Less Than or Equal to Size of Spill	Cumulative Frequency of Spill
1	4	452	0.03
10	40	2 937	0.20
20	80	4 378	0.30
40	150	6 017	0.42
60	230	7 340	0.51
100	380	8 695	0.66
400	1 500	10 866	0.75
700	2 700	11 517	0.80
3 000	11 000	13 097	0.90
5 000	19 000	13 740	0.95
8 000	30 000	14 247	0.98
15 000	57 000	14 475	1.00

Source: USDOT 1986

C.5.2.2 Frequency of a Spill

In addition to estimating the quantities of wastes that could be spilled into Twenty Mile Creek, it was necessary to estimate the frequency with which such spills could be expected to occur. The method used to estimate spill frequency was similar to that described previously in Section C.5.1.2.

The bridge crossing Twenty Mile Creek is located in the fourth link of the local access route to the OWMC facility. (The links were presented previously in Table C-37). Considering both accident-related and non-accident related (i.e., equipment failure) spills, the spill frequency for this link is given by M.M. Dillon (1985):

$$S_l = (L_l)(A_{fl})(N)(f_{tg})(f_{sa}) + (L_l)(N)(f_{sn})$$

Where:

S_l = truck accident- and non accident-related spill frequency for link l, a^{-1}

L_l = length of link l, km

A_{fl} = general vehicle accident frequency on link l, accidents per vehicle per km

N = number of one-way truck deliveries of special waste to the OWMC facility per year

f_{tg} = ratio of accident frequency for trucks to the general vehicle accident frequency

f_{sa} = number of spills per truck accident, and

f_{sn} = frequency of truck spills from non-accident causes, spills per vehicle per km

As shown in Table C-37, the following data applies to the link of the local access route containing the bridge over Twenty Mile Creek:

$L_1 = 3.5$ km

$A_{f1} = 2.77 \times 10^{-6}$ accidents/vehicle per km

The following assumptions were made to estimate the frequency of accident- and non accident-related truck spills along the link:

- N, the number of special waste deliveries by truck will be 6 550 per year (Monenco 1987)
- f_{tg}, the ratio of accident frequency for trucks to the accident frequency for general vehicles is 1.37 (M.M. Dillon 1986)
- f_{sa}, the number of spills per truck accident is approximately 0.196 (Pilorusso 1983)
- f_{sn}, the frequency of truck spills due to equipment failure is 1×10^{-6} spills per vehicle per km (M.M. Dillon 1986)

Based on the above information, the frequency of all truck spills (of any size) occurring along the link of the local access route intersecting Twenty Mile Creek was estimated to be 0.04 spills per year. The fraction of these spills which actually occur on the bridge can be estimated from the ratio of the bridge length to the total link length:

$$S_b = (L_b/L_1)(S_1)$$

Where:

S_b = frequency of spill on bridge, yr^{-1}

S_l = frequency of spill on link = 0.04 yr^{-1}

L_b = length of bridge = 200 m

L_l = length of link = 3 500 m

The estimated frequency of spills of any size on the bridge is thus:

$$S_b = (0.04\ yr^{-1})(200\ m)/(3\ 500\ m)$$

$$S_b = 0.00228\ yr^{-1}$$

As indicated in Section C.5.2.1, three different spill sizes were chosen for the present analysis. In order to determine the estimated frequency of spills in each size category, the base frequency of all spills on the bridge (S_b given above) were multiplied by the fraction of spill incidents resulting in releases greater than the chosen size (see Table C-13). These calculations are summarized in Table C-39. As shown in this table, spills of 230 liters, 1 500 liters, and 19 000 liters were estimated to occur (on average) once in 875 years, 1 750 years, and 8 750 years, respectively.

At the ultimate facility capacity of 300 000 t/a, the estimated frequency of spills into Twenty Mile Creek would double. Thus spills of 230 liters, 1 500 liters, and 19 000 liters were estimated to occur once in 440 years, 875 years, and 4 400 years, respectively.

TABLE C-39

FREQUENCY OF SPECIFIC SIZE SPILL OCCURRING OVER TWENTY MILE CREEK

Size of Spill (1)	Frequency of a Spill Occurring Over Twenty Mile Creek (S_b)	Frequency of Specific Size Spill Occurring	Frequency Size Spill Occurring Over Twenty Mile Creek	Recurrence Interval (Years) of Specific Size Spill Occurring Over Twenty Mile Creek
230	0.00228	0.50	0.00114	875
1 500	0.00228	0.25	0.00057	1 750
19 000	0.00228	0.05	0.000114	8 750

It should be noted that 25 percent of the special waste to be delivered to the proposed OWMC Facility will be in a non-pumpable state (Dillon 1985). Therefore, the frequency of a spill of pumpable waste into Twenty Mile Creek would be somewhat less than calculated in this section. The frequency for a spill of any type of special waste will be used in the analysis, however, since spills of non-pumpable waste may pose some level of acute risk.

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APPENDIX D

TRANSPORT AND FATE OF CONTAMINANTS RELEASED TO THE ATMOSPHERE

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APPENDIX D

TRANSPORT AND FATE OF CONTAMINANTS RELEASED TO THE ATMOSPHERE

D.1 INTRODUCTION

Operation of the proposed OWMC facility will result in the release of potentially hazardous contaminants to the off-site environment. In order to assess the level of risk associated with these releases, it is necessary to estimate the resulting chemical concentrations in the various environmental media and in potential sources of food.

This appendix presents the environmental transport and fate models used to estimate these off-site contaminant concentrations. The models are applied to both routine emissions during normal facility operations and releases which may be caused by upsets and accidents. Chemical concentrations in the following media are calculated:

- Air
- Soil
- Cisterns

- Ponds
- The Welland River
- Plants
- Meat, Milk, and Eggs

Values for the physical and chemical properties of organic contaminants addressed in the transport and fate models are listed in Table D-41 at the end of this appendix.

D.2 ESTIMATION OF CHEMICAL CONCENTRATIONS IN AIR

Chemicals released to the atmosphere during operation of the proposed OWMC facility will be dispersed as they move from the emission source. Resulting chemical concentrations in air can be calculated using the following equation:

$$C_a = Q\ D_s$$

Where:

C_a = Concentration in air, $\mu g/m^3$

Q = Emission rate, g/s

D_s = Dispersion factor, $(\mu g/m^3)/(g/s)$

The size of the dispersion factor, and thus the chemical concentration in air, is dependent upon various parameters, including the elevation from which the release occurs, wind speed, and atmospheric stability. The methodology used in the Phase 4B risk assessment to calculate chemical concentrations resulting from both routine and upset facility emissions and from transportation spills is described in this section. For each type of emission source, the dispersion model used and the assumptions relating to elevation of release, averaging time, and dispersion factors are described.

D.2.1 CHEMICAL CONCENTRATIONS IN AIR - ROUTINE EMISSIONS

Routine emissions to the atmosphere are expected from the incinerator stack and the evaporation unit of the physical/ chemical treatment plant. Fugitive releases from various locations at the facility, such as the organic waste storage area, will also occur.

In the Phase 4B risk assessment, long-term modeling of contaminant concentrations resulting from routine emissions was conducted by the Ontario Research Foundation (ORF) using the United States Environmental Protection Agency's Industrial Source Complex (ISC) Model (see ORF 1987 for a discussion of the ISC Model). The ISC Gaussian plume dispersion model calculates contaminant concentrations at an array of potential receptor locations for a range of meteorological parameters, and the results are weighted according to the frequency of occurrence of these parameters over a specified period of time.

Meteorological conditions based on data collected at the preferred site were used as input to the model for calculating long-term ambient air concentrations.

As previously noted, the height from which an emission occurs will influence the extent of contaminant dispersion in air. Consistent with the facility design (Monenco 1985, 1986), the following elevations were assumed for estimating the extent of dispersion for each emission source:

- Incinerator: 60 meters;
- Evaporator: 45 meters;
- Fugitive releases: 12 meters for emissions vented from the physical/chemical treatment plant; 4 meters for emissions at or near ground level.

Different time-averaging periods were used in applying the ISC model to estimate atmospheric dispersion depending on the duration of the emissions, the exposure pathway, and the chemical under consideration. The use of different time-averaging periods is summarized below:

- For evaluating inhalation exposures to carcinogens, the total lifetime dose was used in calculating risk. Therefore, maximum annually-averaged dispersion factors, corresponding to maximum annual concentrations in air, were applied.
- For evaluating inhalation exposures to non-carcinogens, risk was evaluated through a comparison with an acceptable daily dose. It can be conservatively assumed that a two-week exposure to a non-carcinogen in excess of the acceptable daily dose may result in the manifestation of a toxic effect.

Therefore, chemical concentrations in air based on maximum two-week averaged dispersion factors were used to estimate the level of inhalation exposure to non-carcinogens. Two-week average dispersion factors are greater than those that are annually averaged, resulting in higher predicted concentrations in air.

- For evaluating exposures to both carcinogens and non-carcinogens through ingestion or dermal absorption of contaminants in soil, water or sources of food, maximum annually-averaged dispersion factors were applied.

The dispersion factors used in the Phase 4B risk assessment to evaluate incinerator, evaporator, and fugitive emissions are presented in Table D-1. The factors are expressed as estimated chemical concentrations in ambient air for a unit emission rate. Using these dispersion factors, the long-term ambient air concentrations corresponding to incinerator, evaporator, and fugitive emissions were calculated and are presented in Tables D-2, D-3, and D-4, respectively.

D.2.2 CHEMICAL CONCENTRATIONS IN AIR - SHORT-TERM RELEASES

D.2.2.1 On-Site Upsets and Accidents

Dispersion factors for estimating the atmospheric dispersion of chemicals released as the result of upsets and accidents at the OWMC facility were provided by the Ontario Research Foundation (ORF 1987). In the models, fumigation conditions were assumed for releases from the incinerator stack; a ground level inversion was assumed for the other short-term emissions.

TABLE D-1

LONG-TERM DISPERSION FACTORS

Emission Source	Average Annual $(mg/m^3)/(g/s)$	Maximum 2-week $(mg/m^3)/(g/s)$
Incinerator	6.4×10^{-5}	6.12×10^{-4}
Evaporator	4.7×10^{-4}	2.21×10^{-3}
Fugitive		
- physical/chemical	1.5×10^{-3}	1.25×10^{-2}
- waste loading and storage	3.0×10^{-3}	2.27×10^{-2}

Source: Ontario Research Foundation (ORF 1987). Dispersion factors used in this analysis were taken from a draft version of the ORF Atmospheric Conditions Report. Final factors differ slightly from those in the table, but would not effect the general conclusions of the risk assessment.

TABLE D-2

LONG-TERM AMBIENT AIR CONCENTRATIONS INCINERATOR EMISSIONS

CHEMICAL CONSTITUENT	INCINERATOR ANNUAL CONCENTRATION (mg/m^3)	INCINERATOR MAX 2 WEEK CONCENTRATION (mg/m^3)
ACETONE	7.93E-07	7.58E-06
ACRYLONITRILE	4.94E-09	4.72E-08
BENZENE	1.65E-07	1.58E-06
BENZO(A)ANTHRACENE	3.73E-10	3.57E-09
BENZO(A)PYRENE	1.49E-10	1.43E-09
BROMODICHLOROMETHANE	5.63E-09	5.38E-08
BROMOFORM	4.35E-10	4.16E-09
BUTADIENE,1,3-	4.40E-10	4.21E-09
CARBON TETRACHLORIDE	2.50E-08	2.39E-07
CHLOROBENZENE	1.79E-09	1.71E-08
CHLOROFORM	2.59E-07	2.48E-06
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.99E-13	1.91E-12
DIOXIN,2,3,7,8-EQ(MOE)	4.15E-12	3.96E-11
ETHANOL	1.16E-06	1.11E-05
ETHYLENE GLYCOL	1.47E-06	1.40E-05
ETHYLENE OXIDE	3.76E-10	3.59E-09
FORMALDEHYDE	1.33E-08	1.27E-07
HEXACHLOROBENZENE	2.12E-10	2.03E-09
HYDRAZINE	1.23E-12	1.18E-11
METHYL ETHYL KETONE	2.15E-07	2.05E-06
METHYLENE CHLORIDE	4.16E-07	3.98E-06
NAPHTHALENE	1.71E-08	1.64E-07
NITROPHENOL,o-	1.23E-08	1.17E-07
PCB	5.32E-10	5.09E-09
PETROLEUM DISTILLATES	1.28E-06	1.23E-05
PHENOL	4.35E-09	4.16E-08
PYRIDINE	2.47E-08	2.36E-07
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	6.22E-07	5.95E-06
TETRAETHYL LEAD	9.84E-11	9.41E-10
TOLUENE	1.12E-06	1.07E-05
TRICHLOROETHANE,1,1,1-	4.32E-07	4.13E-06
TRICHLOROETHYLENE	5.52E-07	5.28E-06
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00
XYLENE	5.63E-07	5.38E-06
ARSENIC	1.92E-09	1.83E-08
BERYLLIUM	1.30E-11	1.25E-10
CADMIUM	2.89E-08	2.76E-07
CHROMIUM (VI)	6.85E-08	6.55E-07
COPPER	1.61E-07	1.54E-06
LEAD	9.72E-07	9.29E-06
MERCURY	3.32E-08	3.18E-07
NICKEL	1.94E-08	1.86E-07
ZINC	2.28E-06	2.18E-05
Acid gases ----------		
HYDROGEN CHLORIDE	6.11E-06	5.84E-05
HYDROGEN FLUORIDE	4.16E-07	3.98E-06
NITROGEN OXIDES	8.13E-05	7.77E-04
SULFUR DIOXIDE	6.10E-05	5.83E-04

TABLE D-3

LONG-TERM AMBIENT AIR CONCENTRATIONS
EVAPORATOR EMISSIONS

CHEMICAL CONSTITUENT	EVAPORATOR ANNUAL CONCENTRATION (mg/m^3)	EVAPORATOR MAX 2 WEEK CONCENTRATION (mg/m^3)
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	6.58E-08	3.09E-07
BENZENE	1.27E-08	5.96E-08
BENZO(A)ANTHRACENE	1.22E-11	5.74E-11
BENZO(A)PYRENE	3.29E-11	1.54E-10
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	3.20E-09	1.50E-08
CARBON TETRACHLORIDE	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	8.46E-07	3.97E-06
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00
FORMALDEHYDE	3.05E-10	1.43E-09
HEXACHLOROBENZENE	0.00E+00	0.00E+00
HYDRAZINE	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00
NAPHTHALENE	8.46E-07	3.97E-06
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	1.27E-11	5.96E-11
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	9.40E-09	4.41E-08
PYRIDINE	0.00E+00	0.00E+00
STYRENE	1.22E-08	5.74E-08
TETRACHLOROETHYLENE	4.28E-08	2.01E-07
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	2.26E-07	1.06E-06
TRICHLOROETHANE,1,1,1-	8.46E-07	3.97E-06
TRICHLOROETHYLENE	2.16E-08	1.01E-07
TRICHLOROTRIFLUOROETHANE	7.52E-07	3.53E-06
XYLENE	4.28E-08	2.01E-07
ARSENIC	1.55E-12	7.28E-12
BERYLLIUM	0.00E+00	0.00E+00
CADMIUM	3.10E-14	1.46E-13
CHROMIUM (VI)	1.55E-12	7.28E-12
COPPER	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00
NICKEL	1.55E-12	7.28E-12
ZINC	0.00E+00	0.00E+00

TABLE D-4

LONG-TERM AMBIENT AIR CONCENTRATIONS
FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	ANNUAL CONC (mg/m^3)	MAX 2 WEEK CONC (mg/m^3)
ACETONE	4.25E-05	3.22E-04
ACRYLONITRILE	4.41E-07	3.54E-06
BENZENE	1.73E-06	1.31E-05
BENZO(A)ANTHRACENE	1.95E-18	1.49E-17
BENZO(A)PYRENE	7.76E-19	5.93E-18
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	6.73E-07	5.53E-06
CARBON TETRACHLORIDE	7.97E-07	6.03E-06
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	1.98E-06	1.50E-05
DICHLORODIFLUOROMETHANE	2.18E-06	1.82E-05
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	2.94E-05	2.22E-04
ETHYLENE GLYCOL	1.83E-06	1.38E-05
ETHYLENE OXIDE	7.79E-09	5.89E-08
FORMALDEHYDE	2.23E-05	1.75E-04
HEXACHLOROBENZENE	2.82E-15	2.14E-14
HYDRAZINE	2.39E-11	1.81E-10
METHYL ETHYL KETONE	6.71E-06	5.08E-05
METHYLENE CHLORIDE	4.01E-05	3.04E-04
NAPHTHALENE	5.97E-11	4.97E-10
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	5.32E-11	4.03E-10
PETROLEUM DISTILLATES	2.03E-05	1.53E-04
PHENOL	6.82E-08	5.68E-07
PYRIDINE	4.45E-07	3.37E-06
STYRENE	1.65E-08	1.38E-07
TETRACHLOROETHYLENE	1.10E-05	8.36E-05
TETRAETHYL LEAD	1.49E-09	1.13E-08
TOLUENE	2.97E-05	2.31E-04
TRICHLOROETHANE,1,1,1-	1.29E-05	9.78E-05
TRICHLOROETHYLENE	1.49E-05	1.13E-04
TRICHLOROTRIFLUOROETHANE	3.41E-07	2.84E-06
XYLENE	9.07E-06	6.87E-05
HYDROGEN CYANIDE	2.79E-09	2.18E-08
HYDROGEN SULFIDE	6.86E-08	5.19E-07

During a ground level inversion, a layer of relatively stable air is formed at or close to ground level; emissions into that layer will be poorly dispersed due to a lack of turbulence. Fumigation conditions are those that exist when emissions are initially released into an inversion layer aloft. Emissions can later be dispersed to ground level in relatively high concentrations as an unstable layer, growing upwards from the ground, intersects with the plume aloft. These conditions represent the "worst-case" in estimating off-site short-term chemical concentrations in air.

Consistent with the facility design (Monenco 1985, 1986), the following elevations were assumed for calculating dispersion factors for each emission source:

- Short-term emissions from the incinerator: 60 meters;
- Short-term emissions from the physical/chemical plant: 12 meters;
- Waste unloading and storage: 4 meters.

In applying the dispersion models, different time-averaging periods were used to represent acute exposures depending upon the duration of the emission:

- For evaluating inhalation exposures associated with on-site upset and accident events, maximum one hour dispersion factors were used to calculate maximum one hour air concentrations. These dispersion factors were used for predicting short-term air concentrations associated with the mixing of incompatible wastes, a tank farm fire, the treatment cyanide bearing wastes, and sulfide treatment of wastes containing heavy metals.

- A maximum two-minute air concentration was calculated to assess the effects of an incinerator scrubber failure. The emission from an incinerator scrubber failure can be considered of short duration. As a conservative approach, the release was modeled as a continuous release of thirty second duration (see Appendix C, Section C.4.3).

The maximum one hour and maximum two-minute dispersion factors for on-site upsets and accidents are presented in Table D-5. Using these dispersion factors, the short-term ambient air concentrations corresponding to the on-site upsets and accidents considered were calculated. These concentrations are presented in Table D-6.

D.2.2.2 Transportation Spill onto the Roadway

To predict chemical concentrations in air following the spill of a volatile liquid (methylene chloride) onto a roadway, atmospheric dispersion was estimated using a Gaussian plume model for a ground level source (Turner 1970):

$$x = \frac{Q}{(\pi)(\sigma_y)(\sigma_z)(u)}$$

Where:

x = concentration in the air, g/m^3

Q = emission rate, g/s

σ_y = dispersion coefficient in the lateral direction, m

σ_z = dispersion coefficient in the vertical direction, m

u = windspeed, m/s

TABLE D-5

SHORT-TERM DISPERSION FACTORS

Emission Source	Maximum One Hour $(mg/m^3)/(g/s)$	Maximum Two-Minute $(mg/m^3)/(g/s)$
Incinerator	3.45×10^{-2}	6.9×10^{-2}
Fugitive		
- phys/chem	0.550	1.10
- waste loading and storage	0.796	1.59

Source: Ontario Research Foundation (ORF) 1987. (Maximum two minute disperion factors estimated by ORF from maximum one hour factors.)

TABLE D-6

SHORT-TERM AIR CONCENTRATIONS ASSOCIATED WITH ON-SITE UPSETS AND ACCIDENTS

Event	Surrogate Chemical	Maximum One-Hour Air Concentration (mg/m^3)	Maximum Two-Minute Air Concentration (mg/m^3)
Tank Farm Fire	Methylene Chloride	1 060	--
Mixing of Incompatible Wastes	Hydrogen Sulfide	0.25	--
Incinerator Scrubber Failure	Hydrogen Chloride	--	5.80
	Hydrogen Fluoride	--	0.13
	Nitrogen Oxides	--	0.13
	Sulfur Dioxide	--	4.50
Cyanide Treatment - Uncontrolled pH	Hydrogen Cyanide	1.7×10^{-4}	--
Sulfide Treatment - Uncontrolled pH	Hydrogen Sulfide	7.2×10^{-3}	--

Estimated using the emission estimates summarized in Table C-36 and the dispersion factors reported in Table D-5.

This model is designed for application to a point source emission. To apply it to a pool of liquid, a correction was required to approximate the area source as a point source at some distance upwind from the actual spill site. This correction, referred to as the calculation of a virtual point, was estimated by the method of Turner (1970).

In applying the Gaussian plume model to the spill scenario, it was assumed that the topography in the vicinity of the spill is essentially flat, and that there are no major perturbations introduced by the proximity of large buildings or other topographic irregularities. The atmospheric conditions assumed for dispersion were stability class F and a wind speed of 1 m/s. Class F reflects the most stable condition and thus accounts for the least amount of chemical dispersion (see Gartner Lee Associates [1982] for discussion of atmospheric stability classes). Consistent with the approach of using conservative assumptions throughout the risk assessment, F stability was considered appropriate for modeling atmospheric dispersion.

An important component of the dispersion estimate was the choice of dispersion coefficients. These coefficients depend on the distance from the emission source, and are specific to the atmospheric stability class. The use of such dispersion coefficients in a Gaussian model is generally undertaken at a distance of 100 meters or more, since at shorter distances the behavior of the plume may be significantly influenced by mechanical mixing, particularly for a heavy vapor such as methylene chloride.

In the risk assessment, exposures were evaluated at a distance of 20 meters from the spill site. Therefore, extrapolations were required to apply the Gaussian model. This limitation was recognized in predicting atmospheric concentrations using an extrapolated value (2.8) for the lateral dispersion coefficient and a value of unity for the vertical dispersion coefficient. The methylene chloride concentration in air calculated using this methodology is approximately 18.4 g/m^3.

D.3 ESTIMATION OF CHEMICAL CONCENTRATIONS IN SOIL

Chemicals emitted to the atmosphere from the OWMC facility may deposit onto and accumulate in surface soils. The estimation of chemical concentrations in soil involved the following steps:

- Estimation of the rate of chemical deposition onto soil;
- Estimation of resulting chemical concentrations in surface soil.

These steps are described in this section.

D.3.1 DEPOSITION

Chemicals in the atmosphere can come into contact with ground surfaces through both wet and dry deposition processes. Dry deposition describes the contact of atmospheric particles and gases with a surface as a result of atmospheric turbulence and molecular motion. The wet deposition process involves the washout of chemicals from the atmosphere by precipitation. Both wet and dry processes were considered in calculating the total chemical deposition rate onto soil.

D.3.1.1 Dry Deposition

Dry deposition involves the contact of small particles and gases in the atmosphere with available surfaces such as soil. Upon contact, the particle or gas may adhere to the surficial site through adsorption or other effects. The dry deposition rate has been defined by Hanna et al. (1982):

$$d_{dry} = C\ V_d$$

Where:

d_{dry} = dry deposition rate, $(\mu g/m^2)/s$

C = chemical concentration in air, $\mu g/m^3$

V_d = deposition velocity, m/s

The magnitude of the deposition velocity is a function of particle size and/or certain chemical properties, such as reactivity. Estimates of the deposition velocities used in the present analysis for the chemicals of concern have been developed by the Ontario Research Foundation (ORF 1987), and are presented in Table D-7.

D.3.1.2 Wet Deposition

During precipitation, chemicals are removed from the atmosphere by equilibrium partitioning to raindrops. This results in the wet deposition of chemicals onto soil. It has been suggested that equilibrium between gas and water droplets is established after a fall of approximately ten meters (Slinn and Slinn 1981). Such chemical partitioning between water and air is described in a "washout ratio." This ratio varies depending upon the nature of the chemical and whether it is in the form of a gas, colloid, or particle.

The wet deposition rate can be calculated using the following equation (Hanna et al. 1982):

$$d_{wet} = C\ P\ W\ \frac{D_W}{D_A}$$

Where:

d_{wet} = wet deposition rate, $(\mu g/m^2)/s$

P = annual average precipitation rate, m/s

D_W = density of water, $\mu g/m^3$

D_A = density of air, $\mu g/m^3$

W = washout ratio, dimensionless

C = concentration of chemical in the air, $\mu g/m^3$

TABLE D-7

DEPOSITION VELOCITIES

Chemical Constituent	Deposition Velocity V_d (m/s)
PCDD/PCDF	0.0025
PCBs	0.005
All other organics	0.01
Arsenic	0.0022
Beryllium	0.01
Cadmium	0.0045
Chromium (VI)	0.005
Copper	0.005
Lead	0.003
Mercury	0.01
Nickel	0.0045
Zinc	0.0062

Source: Ontario Research Foundation (ORF 1987)

A washout ratio of 50 was assumed for all organic chemicals; a ratio of 500 was assumed for all inorganic chemicals (ORF 1987).

D.3.1.3 Total Deposition Rate

The total deposition rate was calculated as the sum of the dry and wet deposition rates using the following equation:

$$d = d_{dry} + d_{wet}, \text{ or}$$

$$d = C\,[V_d + P\,W\,\frac{D_W}{D_A}]$$

Where:

d = total chemical deposition rate, $(\mu g/m^2)/s$

d_{dry} = dry deposition rate, $(\mu g/m^2)/s$

d_{wet} = wet deposition rate, $(\mu g/m^2)/s$

C = chemical concentration in air, $\mu g/m^3$

V_d = deposition velocity, m/s

P = average annual precipitation, m/s

D_W = density of water, $\mu g/m^3$

D_A = density of air, $\mu g/m^3$

W = washout ratio, dimensionless

In the risk assessment, the following assumptions were made:

P = 2.79×10^{-8} m/s (Dillon 1986)

D_W = $1\ g/cm^3$

D_A = $1.2 \times 10^{-3}\ g/cm^3$

By applying this equation to the chemical concentrations in air presented in Tables D-2 through D-4, deposition rates onto soil were calculated. The deposition rates calculated for the constituents of concern for the incinerator, evaporator, and fugitive emissions are presented in Tables D-8 through D-10.

D.3.2 CHEMICAL CONCENTRATIONS IN SOIL

Chemicals deposited onto the ground may accumulate in the surface soils over time. Processes such as runoff, leaching to greater depths, degradation by biological microorganisms, volatilization, uptake by plants, and reentrainment to the atmosphere may contribute to the removal of a chemical from these surface soils.

The extent to which chemicals accumulate in soils can be estimated using the following equation (Travis et al. 1983):

$$C_s = \frac{d[1 - e^{-k_{st}t}]}{10 \rho z k_{st}}$$

Where:

C_s = concentration of chemical in soil, μg/kg

d = ground-level deposition rate, μg/m^2-s

ρ = soil bulk density, g/cm^3

z = mixing depth, cm

k_{st} = total rate constant for chemical losses from soil, s^{-1}

t = time during which deposition occurs, s

10 = unit conversion factor

TABLE D-8

DEPOSITION RATES TO SOIL INCINERATOR EMISSIONS

CONTAMINANT	DEPOSITION RATE (ug/m^2/s)
ACETONE	8.85E-06
ACRYLONITRILE	5.51E-08
BENZENE	1.85E-06
BENZO(A)ANTHRACENE	4.17E-09
BENZO(A)PYRENE	1.66E-09
BROMODICHLOROMETHANE	6.28E-08
BROMOFORM	4.35E-09
BUTADIENE,1,3-	4.91E-09
CARBON TETRACHLORIDE	2.79E-07
CHLOROBENZENE	2.00E-08
CHLOROFORM	2.89E-06
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	7.31E-13
DIOXIN,2,3,7,8-EQ(MOE)	1.52E-11
ETHANOL	1.30E-05
ETHYLENE GLYCOL	1.64E-05
ETHYLENE OXIDE	4.20E-09
FORMALDEHYDE	1.48E-07
HEXACHLOROBENZENE	2.37E-09
HYDRAZINE	1.37E-11
METHYL ETHYL KETONE	2.40E-06
METHYLENE CHLORIDE	4.65E-06
NAPHTHALENE	1.91E-07
NITROPHENOL,o-	1.37E-07
PCB	3.28E-09
PETROLEUM DISTILLATES	1.43E-05
PHENOL	4.85E-08
PYRIDINE	2.75E-07
STYRENE	0.00E+00
TETRACHLOROETHYLENE	6.94E-06
TETRAETHYL LEAD	1.10E-09
TOLUENE	1.25E-05
TRICHLOROETHANE,1,1,1-	4.83E-06
TRICHLOROETHYLENE	6.17E-06
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	6.28E-06
ARSENIC	2.65E-08
BERYLLIUM	2.82E-10
CADMIUM	4.66E-07
CHROMIUM (VI)	1.14E-06
COPPER	2.68E-06
LEAD	1.42E-05
MERCURY	7.19E-07
NICKEL	3.14E-07
ZINC	4.06E-05

TABLE D-9

DEPOSITION RATES TO SOIL EVAPORATOR EMISSIONS

CONTAMINANT	DEPOSITION RATE (ug/m^2/s)
ACETONE	0.00E+00
ACRYLONITRILE	7.35E-07
BENZENE	1.42E-07
BENZO(A)ANTHRACENE	1.36E-10
BENZO(A)PYRENE	3.83E-10
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	3.57E-08
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	9.44E-06
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	3.41E-09
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	9.44E-06
NITROPHENOL,o-	0.00E+00
PCB	7.82E-11
PETROLEUM DISTILLATES	0.00E+00
PHENOL	1.05E-07
PYRIDINE	0.00E+00
STYRENE	1.36E-07
TETRACHLOROETHYLENE	4.77E-07
TETRAETHYL LEAD	0.00E+00
TOLUENE	2.52E-06
TRICHLOROETHANE,1,1,1-	9.44E-06
TRICHLOROETHYLENE	2.41E-07
TRICHLOROTRIFLUOROETHAN	9.44E-06
XYLENE	4.77E-07
ARSENIC	2.15E-11
BERYLLIUM	0.00E+00
CADMIUM	5.00E-13
CHROMIUM (VI)	2.58E-11
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	2.50E-11
ZINC	0.00E+00

TABLE D-10

DEPOSITION RATES TO SOIL FUGITIVE EMISSIONS

CONTAMINANT	FUGITIVE DEPOSITION RATE (ug/m^2/s)
ACETONE	4.75E-04
ACRYLONITRILE	5.11E-06
BENZENE	1.93E-05
BENZO(A)ANTHRACENE	3.23E-17
BENZO(A)PYRENE	8.58E-18
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	7.52E-06
CARBON TETRACHLORIDE	8.89E-06
CHLOROBENZENE	0.00E+00
CHLOROFORM	2.21E-05
DICHLORODIFLUOROMETHANE	2.79E-05
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	2.60E-04
ETHYLENE GLYCOL	2.04E-05
ETHYLENE OXIDE	1.26E-08
FORMALDEHYDE	2.49E-04
HEXACHLOROBENZENE	3.05E-14
HYDRAZINE	2.36E-10
METHYL ETHYL KETONE	7.49E-05
METHYLENE CHLORIDE	4.48E-04
NAPHTHALENE	6.66E-10
NITROPHENOL,o-	0.00E+00
PCB	3.20E-10
PETROLEUM DISTILLATES	2.26E-04
PHENOL	5.18E-07
PYRIDINE	5.03E-06
STYRENE	1.85E-07
TETRACHLOROETHYLENE	1.23E-04
TETRAETHYL LEAD	1.67E-08
TOLUENE	3.31E-04
TRICHLOROETHANE,1,1,1-	1.44E-04
TRICHLOROETHYLENE	1.66E-04
TRICHLOROTRIFLUOROETHANE	3.81E-06
XYLENE	1.02E-04

The following assumptions were made in the risk assessment:

ρ = 1.16 g/cm^3 (Ecologistics 1986)
z = 15 cm
t = 2.21 x 10^9 s (70 years)

The calculation of k_{st}, total rate constant for chemical losses from soil, is discussed in the following sections.

D.3.2.1 Volatilization

Chemicals present in soil may be released to the atmosphere as vapors through a process known as volatilization. In the risk assessment, volatile losses from soil were considered only for organic chemicals. Such losses have been considered negligible for all metals, even those considered relatively volatile, e.g., mercury. It was also assumed that volatilization of polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) bound to flyash would not occur. These are conservative assumptions which will tend to overestimate the concentration of chemicals present in soil.

The rate constant for volatilization of organics from soil was calculated using the following equation:

$$k_{sv} = \frac{0.693}{t_{1/2}}$$

Where:

k_{sv} = volatilization rate constant, s^{-1}

$t_{1/2}$ = half-life for soil volatilization, s

The half-life for volatilization, $t_{1/2}$, was calculated using the method of Bomberger et al. (1982):

$$t_{1/2} = \frac{z^2}{4.16\, D_{sa}}$$

Where:

$t_{1/2}$ = the half-life for diffusion of a chemical from the soil, s

z = depth to which the chemical is found, cm

D_{sa} = diffusion coefficient for the chemical in soil air, cm^2/s

The diffusion coefficient for the chemical in soil air was calculated as follows (Bomberger et al. 1982):

$$D_{sa} = \frac{D_a T_a}{\alpha}$$

Where:

D_{sa} = diffusion coefficient for the chemical in soil air, cm^2/s

D_a = diffusion coefficient for the chemical in free air, cm^2/s

T_a = correction for the porosity of the soil, dimensionless

α = correction factor for chemical adsorption on the soil solid and dissolution in the soil water, dimensionless

The correction factors are defined in the following manner:

$$T_a = \frac{\theta_a^{10/3}}{(\theta_a + \theta_w)^2}$$

Where:

θ_a = volume fraction of the soil that is air, dimensionless

θ_w = volume fraction of the soil that is water, dimensionless

and

$$\alpha = \theta_a + \frac{\theta_w}{H} + \frac{\rho K_{oc} F_{oc}}{H}$$

Where:

H = Henry's Law constant, dimensionless

ρ = soil bulk density, g/cm^3

K_{oc} = soil adsorption coefficient based on organic carbon, $(mg/g)/(mg/cm^3)$

F_{oc} = fraction of organic carbon in the soil, g/g

θ_a = volume fraction of the soil that is air, dimensionless

θ_w = volume fraction of the soil that is water, dimensionless

Therefore, the soil volatilization rate constant can be expressed as:

$$k_{sv} = \frac{\dfrac{(4.16 \times 0.693)}{z^2} \dfrac{(D_a \times \theta_a^{10/3})}{(\theta_a + \theta_w)^2}}{\theta_a + \dfrac{\theta_w}{H} + \dfrac{\rho K_{oc} F_{oc}}{H}}$$

In the current analysis the following assumptions were made:

z = 15 cm (assumed mixing depth)

θ_a = 0.12 (Ecologistics 1987)

θ_w = 0.34 (Ecologistics 1987)

ρ = 1.16 g/cm^3 (Ecologistics 1987)

F_{oc} = 0.034 (Ecologistics 1987)

Henry's Law constants (H), diffusion coefficients (D_a), and soil adsorption coefficients (K_{oc}), for the chemicals of concern are presented in Table D-41 at the end of this appendix.

D.3.2.2 Leaching

Chemicals that are soluble in water will leach from the soil and migrate vertically downwards from the mixing zone. The extent of such migration is dependent upon the amount of the precipitation, the soil porosity and the tendency of the chemical to adsorb onto organic carbon in the soil.

The leaching loss rate constant was calculated using a method developed by Baes et al. (1984):

$$k_{sl} = \frac{P - E}{\theta_w \, z \, [1 + (\frac{\rho}{\theta_w} K_d)]}$$

Where:

k_{sl} = leaching loss rate constant, s^{-1}

P = annual average precipitation rate, cm/s

E = annual average evapotranspiration rate, cm/s

θ_w = volumetric water content of the soil, dimensionless

z = mixing depth, cm

ρ = soil bulk density, g/cm^3

K_d = distribution coefficient, ml/g

For organic chemicals, the distribution coefficient (K_d) was defined as follows (Travis et al. 1983):

$$\log K_d = -0.99 + 0.53 (\log K_{ow})$$

Where:

K_d = distribution coefficient, ml/g

K_{ow} = octanol/water partition coefficient, dimensionless

For this analysis, the following assumptions were made:

P = 2.794×10^{-6} cm/s (Dillon 1986)

E = 1.80×10^{-6} cm/s (Dillon 1986)

θ_W = 0.34 (Ecologistics 1987)

z = 15 cm

ρ = 1.16 g/cm^3 (Ecologistics 1987)

K_{ow} values for organic contaminants are shown in Table D-41 at the end of this appendix. Corresponding K_d values are shown in Table D-11. For the metals, K_d values derived by Baes et al. (1984) were adopted. These values are presented in Table D-12.

D.3.2.3 Biodegradation

Biodegradation of chemicals in soils is highly variable. As a result of limitations in available data describing chemical biodegradation in soils, it was conservatively assumed that such losses are negligible in the Phase 4B risk assessment.

TABLE D-11

DISTRIBUTION COEFFICIENTS FOR ORGANICS

Chemical Constituent	K_d
Acetone	0.076
Acrylonitrile	0.14
Benzene	1.38
Benzo(a)anthracene	96.6
Benzo(a)pyrene	167
Bromodichloromethane	1.33
Bromoform	1.87
1,3-Butadiene	1.16
Carbon tetrachloride	3.23
Chlorobenzene	3.27
Chloroform	1.13
Dichlorodifluoromethane	1.29
2,3,7,8-TCDD	334
Ethanol	0.069
Ethylene glycol	0.010
Ethylene oxide	0.071
Formaldehyde	0.046
Hexachlorobenzene	257
Hydrazine	0.019
Methyl ethyl ketone	0.14
Methylene chloride	0.48
Naphthalene	5.67
o-Nitrophenol	0.87
PCB	163
Petroleum distillates	0.23
Phenol	0.61
Pyridine	0.22
Styrene	3.75
Tetrachloroethylene	2.44
Tetraethyl lead	27.0
Toluene	2.73
1,1,1-Trichloroethane	2.19
Trichloroethylene	1.96
Trichlorofluoroethane	6.41
Xylene	4.39

Derived using the method of Travis et al. (1983)

TABLE D-12

DISTRIBUTION COEFFICIENTS FOR METALS

Chemical Constituent	K_d
Arsenic	200
Beryllium	650
Cadmium	65
Chromium	850
Copper	35
Lead	900
Mercury	10
Nickel	150
Zinc	40

Source: Baes et al. (1984)

D.3.2.4 Soil Loss

During storm events, chemicals will be removed from the soil through runoff. Soil loss was calculated on an annually averaged basis using the Universal Soil Loss Equation (Wischmeier and Smith 1978):

$$X = 2.24(R)(K)(LS)(C)(P)(SD)$$

Where:

X = sediment yield per unit area, t/ha/a
R = rain erosivity, dimensionless
K = soil erodibility, t/ha/a
LS = slope and slope length factor, dimensionless
C = cover factor, dimensionless
P = conservation practice, dimensionless
SD = sediment delivery, dimensionless

Inputs to this equation were calculated using data provided by M.M. Dillon Consultants and Engineers, Inc. (Dillon 1986). The following factors were used in the analysis:

R = 100 (Harris 1985), dimensionless

K = 0.21 for clay loam, 3% organic content (Ontario Institute of Pedology (OIP); Harris 1985), t/ha/a

LS = 0.4 for 3% slope, 200 m length (OIP), dimensionless

C = 0.14 for crop rotation consisting of: corn, corn, corn, wheat, hay, hay (OIP; Mills et al. 1985), dimensionless

P = 1 for no conservation practices (OIP; Mills et al. 1985), dimensionless

SD = 0.25 (Agricultural Watershed Studies (AWS) 1978), dimensionless

These assumptions are consistent with the topography, land use, and soil characteristics in the vicinity of the site. The annual sediment yield was calculated as follows:

$$Y = (X)(A)$$

Where:

Y = annual sediment yield, t/a

A = area of the watershed, ha

X = sediment yield per unit area, t/ha/a

Assuming that the watershed area equals 15 700 ha (based on a semicircular drainage basin with a radius of 10 km) the soil loss (Y) was calculated to be 10 340 tons per year (1.03×10^7 kg/a). This compares favorably with 16 000 tons per year calculated based on sediment loadings in the river (calculated based on 331 mg/l sediment concentration in the river as reported by M.M. Dillon (1986) and the average runoff over the area of the watershed.

The soil loss rate constant can be calculated from the annual sediment yield as follows:

$$k_{sr} = \frac{Y}{V}$$

Where:

k_{sr} = runoff rate constant, a^{-1}

Y = annual sediment yield, kg/a

V = mass of soil in the active zone, kg

The mass of soil in the active zone, V, is calculated as:

$$V = 1 \times 10^{6} Az\rho$$

Where:

A = area of watershed, km^2

z = depth of the mixing zone, m

ρ = soil bulk density, kg/m^3

Based on this equation, the mass of soil in the active zone was calculated to be 2.73×10^{10} kg. The resulting total soil loss rate constant was calculated as $3.85 \times 10^{-4}\ a^{-1}$, or $1.22 \times 10^{-11}\ s^{-1}$.

D.3.2.5 Overall Soil Loss Rate Constant

An overall soil loss constant can be calculated by summing the loss constants for volatilization, leaching, and runoff. Therefore,

$$k_{st} = (k_{sv} + k_{sl})(f) + k_{sr}$$

Where:

k_{st} = overall soil loss rate constant, s^{-1}

k_{sv} = volatilization rate constant, s^{-1}

k_{sl} = leaching loss rate constant, s^{-1}

k_{sr} = runoff rate constant, s^{-1}

f = fraction of the year during which losses are assumed to occur, dimensionless

Due to the location of the proposed OWMC facility, the ground is expected to be frozen for a significant portion of the year. During this time, it is unlikely that removal of pollutants from the soil will occur. Therefore, in this analysis, it was conservatively assumed that removal of chemicals from the soil will only occur nine months per year. Therefore, the value of f in the above equation is estimated as 9/12, or 0.75. Because the derivation of soil yield (Y) already accounts for the time the ground is frozen, the correction factor is not applied to the k_{sr}.

The rate constants for soil loss are shown in Table D-13. The corresponding chemical concentrations in soil are presented in Tables D-14, D-15, and D-16 for incinerator, evaporator, and fugitive emissions, respectively.

D.4 ESTIMATION OF CHEMICAL CONCENTRATIONS IN SURFACE WATER AND CISTERNS

Information on water use in the vicinity of the proposed OWMC site indicates that residences use both ponds and cisterns as sources of drinking water. (Cisterns are closed reservoirs, usually underground, used to collect rainwater.) In addition, ponds and the Welland River may also be used for watering livestock. Therefore, chemical concentrations in the following were estimated:

TABLE D-13

RATE CONSTANTS FOR SOIL LOSS

CHEMICAL CONSTITUENT	SOIL VOLATIL RATE CONSTANT Ksv (1/s)	SOIL LEACHING RATE CONSTANT Ksl (1/s)	RUNOFF RATE CONSTANT Ksr (1/s)	TOTAL SOIL LOSS RATE CONSTANT Kst (1/s)
ACETONE	6.55E-09	1.54E-07	1.21E-11	1.20E-07
ACRYLONITRILE	5.27E-08	1.32E-07	1.21E-11	1.38E-07
BENZENE	3.70E-07	3.40E-08	1.21E-11	3.03E-07
BENZO(A)ANTHRACENE	1.45E-14	5.87E-10	1.21E-11	4.52E-10
BENZO(A)PYRENE	2.51E-16	3.40E-10	1.21E-11	2.67E-10
BROMODICHLOROMETHANE	1.61E-07	3.51E-08	1.21E-11	1.47E-07
BROMOFORM	1.88E-08	2.63E-08	1.21E-11	3.38E-08
BUTADIENE,1,3-	2.35E-06	3.91E-08	1.21E-11	1.79E-06
CARBON TETRACHLORIDE	2.99E-07	1.61E-08	1.21E-11	2.36E-07
CHLOROBENZENE	4.23E-08	1.60E-08	1.21E-11	4.37E-08
CHLOROFORM	3.29E-07	3.99E-08	1.21E-11	2.76E-07
DICHLORODIFLUOROMETHANE	3.44E-05	3.58E-08	1.21E-11	2.58E-05
DIOXIN,2,3,7,8-EQ(EPA)	2.55E-14	1.70E-10	1.21E-11	1.40E-10
DIOXIN,2,3,7,8-EQ(MOE)	2.55E-14	1.70E-10	1.21E-11	1.40E-10
ETHANOL	9.16E-10	1.57E-07	1.21E-11	1.18E-07
ETHYLENE GLYCOL	3.44E-15	1.88E-07	1.21E-11	1.41E-07
ETHYLENE OXIDE	1.02E-09	1.56E-07	1.21E-11	1.18E-07
FORMALDEHYDE	1.33E-07	1.68E-07	1.21E-11	2.25E-07
HEXACHLOROBENZENE	1.79E-12	2.21E-10	1.21E-11	1.79E-10
HYDRAZINE	1.04E-11	1.82E-07	1.21E-11	1.37E-07
METHYL ETHYL KETONE	2.05E-08	1.31E-07	1.21E-11	1.14E-07
METHYLENE CHLORIDE	6.89E-07	7.39E-08	1.21E-11	5.72E-07
NAPHTHALENE	1.82E-09	9.53E-09	1.21E-11	8.52E-09
NITROPHENOL,o-	3.83E-10	4.91E-08	1.21E-11	3.71E-08
PCB	1.07E-11	3.49E-10	1.21E-11	2.82E-10
PETROLEUM DISTILLATES	1.41E-06	1.08E-07	1.21E-11	1.14E-06
PHENOL	8.99E-11	6.31E-08	1.21E-11	4.74E-08
PYRIDINE	4.17E-12	1.10E-07	1.21E-11	8.26E-08
STYRENE	6.17E-08	1.41E-08	1.21E-11	5.69E-08
TETRACHLOROETHYLENE	2.01E-07	2.08E-08	1.21E-11	1.67E-07
TETRAETHYL LEAD	5.87E-09	2.08E-09	1.21E-11	5.97E-09
TOLUENE	9.99E-08	1.88E-08	1.21E-11	8.90E-08
TRICHLOROETHANE,1,1,1-	4.49E-07	2.29E-08	1.21E-11	3.54E-07
TRICHLOROETHYLENE	3.31E-07	2.52E-08	1.21E-11	2.67E-07
TRICHLOROTRIFLUOROETHANE	3.25E-06	8.49E-09	1.21E-11	2.44E-06
XYLENE	7.84E-08	1.21E-08	1.21E-11	6.79E-08
ARSENIC		2.84E-10	1.21E-11	2.25E-10
BERYLLIUM		8.74E-11	1.21E-11	7.77E-11
CADMIUM		8.71E-10	1.21E-11	6.65E-10
CHROMIUM (VI)		6.69E-11	1.21E-11	6.23E-11
COPPER		1.61E-09	1.21E-11	1.22E-09
LEAD		6.32E-11	1.21E-11	5.95E-11
MERCURY		5.52E-09	1.21E-11	4.16E-09
NICKEL		3.78E-10	1.21E-11	2.96E-10
ZINC		1.41E-09	1.21E-11	1.07E-09

TABLE D-14

SOIL CONCENTRATIONS - INCINERATOR EMISSIONS

CHEMICAL CONSTITUENT	SOIL CONC (ug/kg)
ACETONE	4.23E-01
ACRYLONITRILE	2.29E-03
BENZENE	3.51E-02
BENZO(A)ANTHRACENE	3.34E-02
BENZO(A)PYRENE	1.59E-02
BROMODICHLOROMETHANE	2.45E-03
BROMOFORM	8.25E-04
BUTADIENE,1,3-	1.57E-05
CARBON TETRACHLORIDE	6.79E-03
CHLOROBENZENE	2.63E-03
CHLOROFORM	6.02E-02
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	7.98E-06
DIOXIN,2,3,7,8-EQ(MOE)	1.66E-04
ETHANOL	6.31E-01
ETHYLENE GLYCOL	6.67E-01
ETHYLENE OXIDE	2.04E-04
FORMALDEHYDE	3.79E-03
HEXACHLOROBENZENE	2.48E-02
HYDRAZINE	5.78E-07
METHYL ETHYL KETONE	1.21E-01
METHYLENE CHLORIDE	4.67E-02
NAPHTHALENE	1.29E-01
NITROPHENOL,o-	2.12E-02
PCB	3.10E-02
PETROLEUM DISTILLATES	7.22E-02
PHENOL	5.88E-03
PYRIDINE	1.92E-02
STYRENE	0.00E+00
TETRACHLOROETHYLENE	2.39E-01
TETRAETHYL LEAD	1.06E-03
TOLUENE	8.05E-01
TRICHLOROETHANE,1,1,1-	7.84E-02
TRICHLOROETHYLENE	1.33E-01
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	5.31E-01
ARSENIC	2.65E-01
BERYLLIUM	3.29E-03
CADMIUM	3.10E+00
CHROMIUM (VI)	1.35E+01
COPPER	1.18E+01
LEAD	1.69E+02
MERCURY	9.95E-01
NICKEL	2.92E+00
ZINC	1.97E+02

TABLE D-15

SOIL CONCENTRATIONS - EVAPORATOR EMISSIONS

CHEMICAL CONSTITUENT	SOIL CONC (ug/kg)
ACETONE	0.00E+00
ACRYLONITRILE	3.05E-02
BENZENE	2.69E-03
BENZO(A)ANTHRACENE	1.09E-03
BENZO(A)PYRENE	3.67E-03
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.14E-04
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	2.23E-03
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	8.68E-05
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	6.37E+00
NITROPHENOL,o-	0.00E+00
PCB	7.39E-04
PETROLEUM DISTILLATES	0.00E+00
PHENOL	1.27E-02
PYRIDINE	0.00E+00
STYRENE	1.38E-02
TETRACHLOROETHYLENE	1.65E-02
TETRAETHYL LEAD	0.00E+00
TOLUENE	1.63E-01
TRICHLOROETHANE,1,1,1-	1.53E-01
TRICHLOROETHYLENE	5.20E-03
TRICHLOROTRIFLUOROETHAN	2.22E-02
XYLENE	4.04E-02
ARSENIC	2.14E-04
BERYLLIUM	0.00E+00
CADMIUM	3.33E-06
CHROMIUM (VI)	3.06E-04
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	2.33E-04
ZINC	0.00E+00

TABLE D-16

SOIL CONCENTRATIONS - FUGITIVE EMISSIONS

FOOD PATHWAYS CHEMICAL CONSTITUENT	FUGITIVE SOIL CONC (ug/kg)
ACETONE	2.27E+01
ACRYLONITRILE	2.05E-01
BENZENE	3.66E-01
BENZO(A)ANTHRACENE	1.74E-10
BENZO(A)PYRENE	8.30E-11
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	2.41E-02
CARBON TETRACHLORIDE	2.16E-01
CHLOROBENZENE	0.00E+00
CHLOROFORM	4.59E-01
DICHLORODIFLUOROMETHANE	5.76E-03
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	1.59E+01
ETHYLENE GLYCOL	8.32E-01
ETHYLENE OXIDE	4.24E-03
FORMALDEHYDE	6.35E+00
HEXACHLOROBENZENE	3.30E-07
HYDRAZINE	1.12E-05
METHYL ETHYL KETONE	3.79E+00
METHYLENE CHLORIDE	4.50E+00
NAPHTHALENE	4.49E-04
NITROPHENOL,o-	0.00E+00
PCB	3.10E-03
PETROLEUM DISTILLATES	1.14E+00
PHENOL	9.23E-02
PYRIDINE	3.46E-01
STYRENE	1.87E-02
TETRACHLOROETHYLENE	4.25E+00
TETRAETHYL LEAD	1.61E-02
TOLUENE	2.14E+01
TRICHLOROETHANE,1,1,1-	2.34E+00
TRICHLOROETHYLENE	3.58E+00
TRICHLOROTRIFLUOROETHANE	8.96E-03
XYLENE	8.57E+00

- Cisterns
- Welland River
- Ponds

The methodologies used to estimate chemical concentrations in these sources of water are discussed in this section.

D.4.1 CHEMICAL CONCENTRATIONS IN CISTERNS

Chemical concentrations in cistern water were assumed to be controlled by one of two different mechanisms, depending upon whether gaseous or particulate emissions were being considered. For gaseous pollutants, Henry's Law describes how a chemical will distribute itself between air and water under ideal conditions (Lyman et al. 1982):

$$C_W = \frac{C_A}{H}$$

Where:

C_W = equilibrium aqueous concentration (dilute) of the chemical, g/cm^3

C_A = equilibrium air concentration of the chemical above the dilute aqueous solution, g/cm^3

H = Henry's Law constant, dimensionless

Henry's Law constants for the chemicals of concern are listed in Table D-41 at the end of this appendix. Chemical concentrations in air were presented in Section D.2.1.

Certain chemicals released from the proposed OWMC facility may be adsorbed onto particulate matter such as flyash. For example, metals emitted from an incinerator will not remain in the vapor phase; they will quickly adsorb to particles or exist as solid oxides. For such contaminants, Henry's Law cannot be used to estimate the concentrations in cistern water. Instead, concentrations were calculated by estimating the mass of material deposited into a volume of water, and subsequently released from the particles by desorption. Neglecting any dilution afforded by the initial contents of the cistern, the long-term concentration of metals in such a cistern can be conservatively estimated using the following formula:

$$C_m = \frac{(d)(f_s)}{P-E}$$

Where:

C_m = concentration in the cistern, g/m^3

d = deposition rate, $g/m^2/a$

P = average annual precipitation, m/a

E = average annual evapotranspiration, m/a

f_s = fraction of contaminant on flyash which enters the aqueous phase, dimensionless

For this analysis, the following assumptions were made:

P = 0.881 m/a (Dillon 1986)

E = 0.569 m/a (Dillon 1986)

f_s = 1.0

As a conservative assumption, it was assumed that the fraction of all metals originally on the flyash that would dissolve in the aqueous phase (f_s) would be one. The particulate deposition approach was used in estimating chemical concentrations for all metals emitted from the incinerator.

The Henry's Law approach was applied for all organic chemicals with the exception of polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF). Since PCDD/PCDF may be strongly adsorbed to flyash emitted from the incinerator, both the Henry's Law and particulate deposition approaches were considered. A flyash-water partition coefficient of 0.09 was adopted as the lowest value reported in the literature (Matsumura and Ward, 1976). Using both methods, the particulate deposition approach was found to result in the highest water concentrations. Consistent with the conservative approach used throughout the risk assessment, this higher value was adopted for estimating the concentrations of PCDD/PCDF in cistern water.

The concentrations of the surrogate chemicals in cistern water for the three emission sources are shown in Tables D-17 through D-19.

D.4.2 CHEMICAL CONCENTRATIONS IN THE WELLAND RIVER

Chemicals released from the OWMC facility can reach the Welland River through two primary pathways: (1) direct deposition onto

TABLE D-17

CISTERN CONCENTRATIONS - INCINERATOR EMISSIONS

CONTAMINANT	CISTERN CONC (mg/l)
ACETONE	6.34E-07
ACRYLONITRILE	1.37E-09
BENZENE	7.35E-10
BENZO(A)ANTHRACENE	8.96E-09
BENZO(A)PYRENE	7.30E-09
BROMODICHLOROMETHANE	5.63E-11
BROMOFORM	1.87E-11
BUTADIENE,1,3-	5.19E-14
CARBON TETRACHLORIDE	2.01E-11
CHLOROBENZENE	1.22E-11
CHLOROFORM	1.73E-09
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	6.65E-12
DIOXIN,2,3,7,8-EQ(MOE)	1.38E-10
ETHANOL	7.97E-06
ETHYLENE GLYCOL	2.55E-01
ETHYLENE OXIDE	2.53E-10
FORMALDEHYDE	1.16E-09
HEXACHLOROBENZENE	7.62E-12
HYDRAZINE	2.28E-09
METHYL ETHYL KETONE	1.43E-07
METHYLENE CHLORIDE	4.65E-09
NAPHTHALENE	9.11E-10
NITROPHENOL,o-	3.90E-08
PCB	4.91E-12
PETROLEUM DISTILLATES	5.38E-12
PHENOL	2.34E-07
PYRIDINE	8.51E-05
STYRENE	0.00E+00
TETRACHLOROETHYLENE	8.60E-10
TETRAETHYL LEAD	3.00E-14
TOLUENE	4.02E-09
TRICHLOROETHANE,1,1,1-	6.15E-10
TRICHLOROETHYLENE	1.41E-09
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	2.56E-09
ARSENIC	2.68E-06
BERYLLIUM	2.85E-08
CADMIUM	4.71E-05
CHROMIUM (VI)	1.15E-04
COPPER	2.71E-04
LEAD	1.44E-03
MERCURY	7.27E-05
NICKEL	3.17E-05
ZINC	4.10E-03

TABLE D-18

CISTERN CONCENTRATIONS - EVAPORATOR EMISSIONS

CONTAMINANT	CISTERN CONC (mg/l)
ACETONE	0.00E+00
ACRYLONITRILE	1.83E-08
BENZENE	5.64E-11
BENZO(A)ANTHRACENE	2.93E-10
BENZO(A)PYRENE	1.68E-09
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	3.78E-13
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	9.68E-12
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	2.65E-11
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	4.50E-08
NITROPHENOL,o-	0.00E+00
PCB	1.17E-13
PETROLEUM DISTILLATES	0.00E+00
PHENOL	5.05E-07
PYRIDINE	0.00E+00
STYRENE	6.52E-11
TETRACHLOROETHYLENE	5.92E-11
TETRAETHYL LEAD	0.00E+00
TOLUENE	8.13E-10
TRICHLOROETHANE,1,1,1-	1.20E-09
TRICHLOROETHYLENE	5.52E-11
TRICHLOROTRIFLUOROETHAN	6.93E-11
XYLENE	1.94E-10
ARSENIC	2.17E-09
BERYLLIUM	0.00E+00
CADMIUM	5.06E-11
CHROMIUM (VI)	2.61E-09
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	2.53E-09
ZINC	0.00E+00

TABLE D-19

CISTERN CONCENTRATIONS - FUGITIVE EMISSIONS

CONTAMINANT	CISTERN CONC (mg/l)
ACETONE	3.40E-05
ACRYLONITRILE	1.22E-07
BENZENE	7.68E-09
BENZO(A)ANTHRACENE	4.67E-17
BENZO(A)PYRENE	3.80E-17
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	7.95E-11
CARBON TETRACHLORIDE	6.40E-10
CHLOROBENZENE	0.00E+00
CHLOROFORM	1.32E-08
DICHLORODIFLUOROMETHANE	2.50E-11
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	2.01E-04
ETHYLENE GLYCOL	3.18E-01
ETHYLENE OXIDE	5.25E-09
FORMALDEHYDE	1.94E-06
HEXACHLOROBENZENE	1.01E-16
HYDRAZINE	4.43E-08
METHYL ETHYL KETONE	4.48E-06
METHYLENE CHLORIDE	4.48E-07
NAPHTHALENE	3.17E-12
NITROPHENOL,o-	0.00E+00
PCB	4.98E-13
PETROLEUM DISTILLATES	8.51E-11
PHENOL	3.67E-06
PYRIDINE	1.53E-03
STYRENE	8.85E-11
TETRACHLOROETHYLENE	1.53E-08
TETRAETHYL LEAD	4.56E-13
TOLUENE	1.07E-07
TRICHLOROETHANE,1,1,1-	1.83E-08
TRICHLOROETHYLENE	3.80E-08
TRICHLOROTRIFLUOROETHANE	2.79E-11
XYLENE	4.12E-08

the water surface, or (2) the influx of contaminated runoff into the river. Since the drainage basin for the Welland River covers a significantly larger surface area than the river itself, only the input from runoff was considered in the risk assessment.

Chemicals emitted to the atmosphere from the OWMC facility may be deposited on the ground surface and accumulate in the surface soils. During periods of rainfall, surface soils within the Welland River drainage basin will be transported to the river. The chemicals were assumed to mix with the water to yield average concentrations in the river.

To estimate long-term concentrations of the chemicals in the Welland River, an annually-averaged, areally-averaged approach was used. Although it is recognized that the use of averaged models may underpredict or overpredict the concentrations at particular times, it was considered that long-term effects on livestock would be adequately represented by annual average concentrations. Deviations from average concentrations may occur during winter conditions, when little inflow of pollutants to the river may occur; during spring snowmelt events, when high concentrations of pollutants may exist; and during severe summer storms, which may also result in high concentrations of pollutants.

The following steps were used to calculate contaminant concentrations in the Welland River:

- Estimation of chemical deposition in the river drainage basin;
- Estimation of average chemical concentration in soil throughout the drainage basin;
- Estimation of chemical concentrations in runoff; and
- Estimation of resulting chemical concentrations in the river.

These steps are described in the following subsections.

D.4.2.1 Deposition

As discussed previously in Section D.3.1, chemicals in the atmosphere may be deposited onto surface soils through wet and dry deposition processes. For estimating chemical concentrations in the Welland River through runoff however, it was recognized that use of the maximum deposition rate throughout the entire drainage basin would significantly overestimate the total amount of chemical deposited. An alternative methodology was therefore developed where it was assumed that the total annual mass of each chemical emitted from the facility was deposited on the ground surface within a ten kilometer radius of the site.

D.4.2.2 Concentration in Soil

To estimate concentrations of chemicals in soil, it was assumed that all chemicals in the surface soils would be in a solid state, adsorbed onto dust particles. Thus, the mechanism for pollutants to enter the river was assumed to be by transport of soil particles in runoff. The concentration of a substance in the river, therefore, would correspond directly to the concentration of that substance on the soil. This in turn would depend on the chemical deposition rate, the volume of soil in the active mixing zone, and the depth of soil lost due to erosion and other removal mechanisms. After a sufficiently long period of plant operation, the system would reach steady state.

The concentration of pollutant on the soil at steady-state was calculated assuming the surface soils behave as a completely mixed flow reactor each year as a result of spring and fall plowing and loss of soil through erosion. The concentration was calculated from the mass balance equation:

$$\frac{dc}{dt} = \frac{m}{V} - \frac{Yc}{V} - k_{sv}c - k_{s1}c$$

Where:

c = concentration of chemical in the soil zone, mg/kg

t = time, a

V = mass of soil, kg

m = deposition rate of chemical, mg/a

k_{sv} = rate constant for soil volatilization, a^{-1}

k_{sl} = rate constant for soil leaching, a^{-1}

Y = yield of soil, kg/a

The mass of the soil (V) was calculated from:

$$V = 1 \times 10^6 Az\rho$$

Where:

A = area of the watershed, km^2

z = soil mixing zone, m

ρ = soil bulk density, kg/m^3

The solution to this mass balance differential equation is:

$$c = \frac{m}{(Y+Vk_{st})} [1-\exp(-t_d(Y/V+k_{st}))]+c_0 \exp(-t_d(Y/V+k_{st}))$$

Where:

c = concentration of chemical in the soil zone, mg/kg

m = deposition rate of chemical, mg/a

Y = yield of soil, kg/a

V = mass of soil, kg

t_d = design life of the facility, a

c_0 = concentration in the soil zone before start-up of the OWMC facility, mg/kg.

k_{st} = total soil loss rate constant ($k_{sl} + k_{sv}$), a^{-1}

For the purposes of the risk assessment, it was assumed that processes contributing to the background concentrations of contaminants in the Welland River are in steady-state and would continue to be in steady-state during operation of the OWMC facility. The equation used to calculate the concentration of the chemical in the soil zone that would ultimately be discharged to the Welland River can then be simplified as follows:

$$c = \frac{m}{(Y+Vk_{st})} [1-\exp(-t_d(Y/V+k_{st}))]$$

Where:

c = concentration of chemical in the soil zone, mg/kg

m = deposition rate of chemical, mg/a

Y = yield of soil, 1.05×10^7 kg/a (calculated in Section D.3.3.4)

V = mass of soil, 2.73×10^{10} kg (calculated in Section D.3.3.4)

k_{st} = total soil loss rate constant, a^{-1}

t_d = design life of facility, a

For this analysis, the following assumptions regarding the characteristics of the watershed were made:

z = 15 cm

t_d = 70 a

A = 157 km^2, based on a semicircular drainage basin with a radius of 10 km

ρ = 1.16 g/cm^3 (Ecologistics 1987)

The total soil loss constant (k_{st}) is the same as that presented in Section D.3.3.4 of this appendix.

D.4.2.3 Concentration of Chemicals in the Runoff

Contaminant concentrations in the runoff are a function of (1) the sediment concentration in the runoff and (2) the concentration of the chemical on the sediment (calculated in section D.4.2.2). The sediment concentration in the runoff was calculated by:

$$c_s = \frac{10^6 (X)(A)}{Vr}$$

Where:

c_s = concentration of sediment in the runoff, mg/l

Vr = annual runoff volume, 1.88×10^{10} l/a (Dillon 1986)

A = area of the watershed, 157 km^2

X = sediment yield per unit area, 6.7×10^4 $kg/km^2/a$

The concentration of pollutant in the runoff can then be calculated by:

$$c_r = c_s c \, (1 \times 10^{-6})$$

Where:

c_r = concentration of chemical in the runoff, mg/l

c_s = concentration of sediment in the runoff, mg/l

c = concentration of chemical in soil mixing zone, mg/kg

D.4.2.4 Concentration of Chemicals in the Welland River

Estimation of predicted pollutant concentrations in the Welland River was undertaken using not only the contribution from the OWMC facility but also the existing concentrations of chemicals in both the river water and sediments. These data were compiled by the Ontario Ministry of the Environment (OMOE 1984) and M.M. Dillon (Dillon 1986), and are presented in Chapter 3. Using these data, the total concentrations of pollutants in the river were calculated using the following equation:

$$c_t = c_i + V_r c_r / q_i$$

Where:

c_t = total concentration in the river, mg/l

c_i = background concentration in the river, mg/l

q_i = total flow of the river, 1.28×10^8 m^3/a (Dillon 1986)

V_r = annual runoff volume, m^3/a

c_r = concentration of chemical in runoff, mg/l

Concentrations of chemicals in the river calculated using this formula are tabulated in Tables D-20 through D-22 for incinerator, evaporator, and fugitive emissions, respectively.

D.4.2.5 Acidification Effects

In addition to the impacts of metals and organic chemicals on river quality, consideration was given to the effects of

TABLE D-20

CONCENTRATION OF CHEMICALS IN THE WELLAND RIVER - INCINERATOR EMISSIONS

CONTAMINANT	INCINERATOR RIVER CONC (mg/l)
ACETONE	3.08E-07
ACRYLONITRILE	1.67E-09
BENZENE	2.55E-08
BENZO(A)ANTHRACENE	2.43E-08
BENZO(A)PYRENE	1.16E-08
BROMODICHLOROMETHANE	1.79E-09
BROMOFORM	6.00E-10
BUTADIENE,1,3-	1.15E-11
CARBON TETRACHLORIDE	4.94E-09
CHLOROBENZENE	1.91E-09
CHLOROFORM	4.38E-08
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.77E-11
DIOXIN,2,3,7,8-EQ(MOE)	3.68E-10
ETHANOL	4.59E-07
ETHYLENE GLYCOL	4.86E-07
ETHYLENE OXIDE	1.49E-10
FORMALDEHYDE	2.76E-09
HEXACHLOROBENZENE	1.80E-08
HYDRAZINE	4.21E-13
METHYL ETHYL KETONE	8.82E-08
METHYLENE CHLORIDE	3.40E-08
NAPHTHALENE	9.39E-08
NITROPHENOL,o-	1.55E-08
PCB	4.08E-08
PETROLEUM DISTILLATES	5.25E-08
PHENOL	1.00E-03
PYRIDINE	1.40E-08
STYRENE	0.00E+00
TETRACHLOROETHYLENE	1.74E-07
TETRAETHYL LEAD	7.70E-10
TOLUENE	5.86E-07
TRICHLOROETHANE,1,1,1-	5.70E-08
TRICHLOROETHYLENE	9.66E-08
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	3.87E-07
ARSENIC	3.00E-03
BERYLLIUM	5.85E-18
CADMIUM	1.00E-03
CHROMIUM (VI)	1.00E-02
COPPER	1.00E-02
LEAD	9.00E-03
MERCURY	4.00E-05
NICKEL	7.00E-03
ZINC	1.70E-02

TABLE D-21

CONCENTRATION OF CHEMICALS IN THE WELLAND RIVER - EVAPORATOR EMISSIONS

CONTAMINANT	RIVER CONC mg/l
ACETONE	0.00E+00
ACRYLONITRILE	3.03E-09
BENZENE	2.67E-10
BENZO(A)ANTHRACENE	1.08E-10
BENZO(A)PYRENE	3.64E-10
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.13E-11
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	2.21E-10
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	8.62E-12
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	6.31E-07
NITROPHENOL,o-	0.00E+00
PCB	1.33E-10
PETROLEUM DISTILLATES	0.00E+00
PHENOL	1.00E-03
PYRIDINE	0.00E+00
STYRENE	1.37E-09
TETRACHLOROETHYLENE	1.63E-09
TETRAETHYL LEAD	0.00E+00
TOLUENE	4.03E-09
TRICHLOROETHANE,1,1,1-	1.52E-07
TRICHLOROETHYLENE	5.15E-10
TRICHLOROTRIFLUOROETHAN	1.96E-08
XYLENE	4.00E-09
ARSENIC	3.00E-03
BERYLLIUM	0.00E+00
CADMIUM	1.00E-03
CHROMIUM (VI)	1.00E-02
COPPER	1.00E-02
LEAD	9.00E-03
MERCURY	4.00E-05
NICKEL	7.00E-03
ZINC	1.70E-02

TABLE D-22

CONCENTRATION OF CHEMICALS IN THE WELLAND RIVER - FUGITIVE EMISSIONS

CONTAMINANT	RIVER CONC mg/l
ACETONE	8.97E-07
ACRYLONITRILE	1.36E-08
BENZENE	1.47E-08
BENZO(A)ANTHRACENE	1.27E-17
BENZO(A)PYRENE	3.56E-18
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.76E-09
CARBON TETRACHLORIDE	8.56E-09
CHLOROBENZENE	0.00E+00
CHLOROFORM	1.82E-08
DICHLORODIFLUOROMETHANE	5.22E-10
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	5.00E-07
ETHYLENE GLYCOL	3.30E-08
ETHYLENE OXIDE	2.42E-11
FORMALDEHYDE	3.39E-07
HEXACHLOROBENZENE	1.26E-14
HYDRAZINE	4.73E-13
METHYL ETHYL KETONE	1.50E-07
METHYLENE CHLORIDE	1.78E-07
NAPHTHALENE	3.55E-11
NITROPHENOL,o-	0.00E+00
PCB	2.17E-10
PETROLEUM DISTILLATES	4.53E-08
PHENOL	1.00E-03
PYRIDINE	1.39E-08
STYRENE	1.48E-09
TETRACHLOROETHYLENE	1.69E-07
TETRAETHYL LEAD	6.36E-10
TOLUENE	1.08E-06
TRICHLOROETHANE,1,1,1-	9.45E-08
TRICHLOROETHYLENE	1.42E-07
TRICHLOROTRIFLUOROETHANE	7.09E-10
XYLENE	3.42E-07

acidification. Acidification of lakes throughout eastern Canada has been attributed to deposition of acid gases emitted from power plants, steel mills, and copper smelters. Since the proposed OWMC facility will produce acid gas emissions, a simple model for the impact of acid gases on the pH of the Welland River was developed. The model was based on the same assumptions used for estimating chemical concentrations in the river with the following exceptions:

- The entire annual emissions were conservatively assumed to be washed to the river each year, i.e., losses due to degradation of buffering in the soil were not considered;
- The buffering capacity of the river was considered by assuming that essentially all of the observed alkalinity naturally occurring in the river is carbonate alkalinity.

The carbonate alkalinity was believed to be appropriate since the bedrock in the region is composed of dolomite limestone. No other mechanism for mitigation of acidity were assumed.

Allowance was made for the existing the background hydrogen ion concentration using the following formula:

$$H_B = 10^{-pH} * 1000$$

Where:

H_B = background hydrogen ion concentration, meq/l

pH = average river pH, 7.6 (Dillon 1986)

The contribution of hydrogen ions from OWMC acid gas emissions was then calculated as:

$$H_{OWMC} = \frac{[HCl]}{36} + \frac{[H_2SO_4]}{49} + \frac{[HNO_3]}{63} + \frac{[HF]}{20}$$

Where:

H_{OWMC} = hydrogen ion concentration contributed by OWMC, meq/l

$[HCl]$ = concentration of HCl in the river, mg/l

$[H_2SO_4]$ = concentration of H_2SO_4 in the river, mg/l

$[HNO_3]$ = concentration of HNO_3 in the river, mg/l

$[HF]$ = concentration of HF in the river, mg/l

The alkalinity after addition of the acid gases is calculated as:

$$ALK_{OWMC} = \frac{ALK_B}{50} - H_{OWMC}$$

Where:

ALK_{OWMC} = alkalinity after addition of OWMC acid gases, meq/l

ALK_B = background alkalinity, meq/l

The final pH is calculated as:

$$pH = -\log\left[\left\{\frac{ALK_B}{ALK_{OWMC}} \times H_B\right\} / 1000\right]$$

Using this methodology, the pH in the river is estimated to drop from 7.6 (current annual average with range of 6.4 to 8.3) to 7.59, so that the acidification effects can be considered negligible.

D.4.3 CHEMICAL CONCENTRATIONS IN PONDS

Chemicals released to the atmosphere from the OWMC facility can reach a pond either through direct deposition onto the surface of the water or through runoff after deposition onto soil. Because of the small size of ponds in the vicinity of the site, both mechanisms were assumed to be significant in the risk assessment. A hypothetical "typical" pond was defined for the purposes of the risk assessment by examining the average characteristics of the ponds within several kilometers of the proposed facility using a topographic map of the site area. Based on this approach, a typical pond was defined as having an area of approximately 2000 square meters and a depth of four meters.

An annually-averaged steady-state model was used in the risk assessment to estimate pollutant concentrations in the hypothetical pond. As discussed in Section D.4.2, such an approach may lead to under- or overestimates of chemical concentrations in the pond at any one time. It is, however, believed to be appropriate for the evaluation of long-term exposures.

The approach used to calculate chemical concentrations in the hypothetical pond consisted of the following steps:

- Examination of the processes through which water enters and leaves the pond;

- Examination of the processes through which sediments enter the pond; and
- Estimation of chemical concentrations in the pond water resulting from both direct chemical deposition and runoff from the surrounding area.

These steps are described in this section.

D.4.3.1 Water Balance for the Hypothetical Pond

In order to estimate the concentrations of chemicals in the hypothetical pond, the processes that determine the quantity of water in the pond were first identified. The volume of water in a pond is a function of precipitation, runoff, evaporation, percolation, and pond usage, as shown in the following water balance:

$$\frac{dV}{dt} = A_p(P-ET) + (Rf) - Q_p - Q_u$$

Where:

dV/dt = change in pond volume over time

P = direct precipitation, m/a

Rf = runoff, m^3/a

ET = evapotranspiration, m/a

Q_p = percolation, m^3/a

Q_u = usage, m^3/a

A_p = area of the pond, m^2

A_i = area of runoff, m^2

In the risk assessment, it was conservatively assumed that any inflow to the typical pond from ground water would be negligible. Furthermore, it was assumed that the volume of water in the pond would not change over time; that is, the pond would remain at steady-state, i.e., $\frac{dV}{dt} = 0$. The quantity of inflow into the pond due to runoff (Ai x P) can therefore be calculated as follows:

$$Rf = Q_p + Q_u - A_p (P-ET)$$

The following values for direct precipitation and evapotranspiration were used in the calculations:

P = 881 mm/a (Dillon 1986)

ET = 569 mm/a (Dillon 1986)

Usage (Q_u) was calculated to be approximately 400 m^3/a based on the average number of people and livestock estimated to take water from nearby ponds, and the mean quantity of water consumed by each user. The pond was assumed to be in use for nine months out of the year, during spring, summer, and fall, and frozen for the remainder of the year. Values utilized in the usage calculations are shown in Table D-23.

Percolation (Q_p) was calculated using a simple Darcy formulation as follows:

$$Q_p = k \vec{i} A_p$$

TABLE D-23

ASSUMED USAGE (Q_u) FOR THE HYPOTHETICAL POND

	Usage Rate l/d	Ref.	Average Users per Pond	Ref.	Total Usage, on Seasonal Basis, m^3/yr
Turkeys	0.8	4	850	1	186.2
Cattle	50.0	5	12	1	164.3
Humans, Potable Use Only	37.9	2	1	3	10.4
Humans, All Uses	170.6	2	1	3	46.7
Total					407.5

Sources:

1. Dillon 1986
2. Steel 1960
3. Gartner Lee 1987
4. Based on NRC 1974
5. Based on Ecologistics 1984; NRC 1974

Where:

Q_p = rate of percolation of water from the pond, m^3/a

k = permeability of the pond bottom, m/a

$\vec{i}$ = hydraulic gradient, m/m

A_p = area of the bottom of the pond, m^2

The following assumptions were made in calculating Q_p:

- The hydraulic gradient ($\vec{i}$) was assumed to be 1 m/m, since it was assumed that the pond would contain water at all times;
- Permeability (k) of silt and loess ranges from 1×10^{-7} to 1×10^{-3} cm/s (Mills et al. 1985). A value of 1×10^{-5} cm/s was used in the model.

From the values of P, ET, Q_p, Q_u, and A_p (2 083 m^2), the total inflow into the pond due to runoff (Rf) was calculated. The area from which runoff would occur can then be calculated from:

$$Ai = Rf/P$$

This value is used in the calculation of sediment loading to the pond described in the following subsection.

D.4.3.2 Sediment Balance for the Hypothetical Pond

The concentration of sediments in the pond was calculated from a material balance for soil in the pond:

$$V\left(\frac{dS}{dt}\right) = Y\,(1 - f_s) - SQ_u$$

Where:

Y = yield of soil, mg/a

V = volume of the pond, 1^3

S = concentration of sediment in pond, mg/l

Q_u = usage rate of water from the pond, l/a

f_s = fraction of material removed by sedimentation, dimensionless

t = time, a

The solution to the equation is:

$$S = \frac{(1-f_s)Y}{Q_u} [1-\exp(-tQ_u/V)]$$

For a particle to settle in a pond within one year, the terminal velocity of the particle must equal:

$$V_{max} = D/t$$

Where:

V_{max} = terminal velocity of a particle, m/s

D = depth of the pond, 4.1 m

t = time, s

From Stokes Law, the diameter of a particle which would have this velocity may be calculated:

$$d = \left[\frac{V_{max}\,(18)\,(\mu)}{g(\rho_s - \rho_w)} \right]^{1/2}$$

V_{max} = terminal velocity of a particle, m/s

ρs = density of the particle, g/cm^3

ρ = density of water, g/cm^3

d = particle diameter, cm

μ = viscosity of water, g/cm-s

g = gravitational acceleration, m/s^2

The following assumptions were made for this analysis:

ρ_s = 2.2 g/cm^3 (average for a clay particle)

ρ_w = 1.0 g/cm^3

μ = 1.4 x 10^{-2} g/(cm-s) (based on annual average temperature of 8.3°C)

g = 9.8 m/s^2

A particle diameter of .0005 mm represented a particle that would just settle in a pond 4.1 meters deep (the average depth assumed for the typical pond) within one year. Particle size distribution analyses of soil samples conducted by Gartner Lee Associates (Gartner Lee 1986) indicated that approximately 70 percent of the particles are larger than 0.001 mm and 30% are smaller. Thus, approximately 70% of the particles reaching the pond will settle within one year.

The Universal Soil Loss Equation (described in Section D.3.3.4) was used to calculate the quantity of soil entering the pond (Y) and the annual yield of soil (Y_s):

X = 2.24 (R)(K)(LS)(C)(P)(SD)

Where:

X = sediment yield per unit area, t/ha/a

R = rain erosivity, 100 (Harris 1985), dimensionless

K = soil erodibility, 0.21 for clay loam, 3% organic content (Ontario Institute of Pedology (OIP); Harris 1985), t/ha/a

LS = slope and slope length factor, .59 for 5% slope, 37 m length (OIP), dimensionless

C = cover factor, 0.004 for meadow, grass & legume mix (OIP, Mills et al. 1985), dimensionless

P = conservation practice, 1 for no conservation practices (OIP, Mills et al. 1985), dimensionless

SD = sediment delivery, 1 (Agricultural Watershed Studies (AWS) 1978), dimensionless

The factors used for slope length and angle, sedimentation factor, and cover factor are different from those used previously in Section D.3.3.4 because of the small area of watershed and the assumption that the area immediately surrounding the pond is not farmed.

From the soil loss equation, a value for the sediment yield per unit area per year (X) was calculated. This value was multiplied by the area from which runoff is predicted to occur (Ai) to estimate the total load of sediment to the hypothetical pond each year.

D.4.3.3 Chemical Concentrations in the Hypothetical Pond

The concentration of chemical in the pond was calculated by material balance:

Amount of chemical in the pond =

amount of chemical deposited either on the pond or in the drainage basin for the pond

- losses due to percolation,

- losses due to usage,

- losses due to volatilization.

This material balance can be expressed mathematically as:

$$V(\frac{dc_T}{dt}) = d - c_d Q_p - c_T Q_u - k_v c_d V$$

Where:

V = volume of the pond, l

$(\frac{dc_T}{dt})$ = the change in contaminant concentrations over time, mg/a

c_T = total concentration of pollutants in the pond, mg/l

t = time, a

d = deposition rate of chemical over the area of the pond and the area from which runoff would occur, mg/a

c_d = concentration of dissolved chemical in the pond, mg/l

Q_p = rate of percolation of water from the pond, l/a

Q_u = usage rate of water from the pond, l/a

k_v = volatilization rate constant, a^{-1}

The solution to the equation is:

$$c_T = d/(V \times [(Q_p/[V(1 + K_dS)]) + Q_u/V + (k_v/[1 + K_dS])]) \times [1 - \exp(-t \times [(Q_p/[V \times (1 + K_dS)]) + Q_u/V + (k_v/[1 + K_dS])])]$$

Where:

K_d = the adsorption equilibrium constant

S = the concentration of sediments in the pond, mg/l

d = deposition rate of chemical over the area of the pond and the area from which runoff would occur, g/a

V = volume of pond, m^3

Q_p = rate of percolation of water from the pond, m^3/a

Q_u = usage rate of water from pond, m^3/a

k_v = volatilization rate constant, a^{-1}

t = time, a

As discussed in Section D.3.1, chemicals in the atmosphere will be deposited through wet and dry deposition processes. For this analysis, it was assumed that the hypothetical "typical" pond was located at the point of maximum deposition for each emission source. Thus, maximum annual deposition rates calculated in Section D.3.1 were used in this analysis.

Chemical volatilization from surface water was estimated using the following table developed by Mills et al. (1985):

H (dimensionless)	K_v (1/d)
41.6	4.8
4.2	4.8
4.2×10^{-1}	4.7
4.2×10^{-2}	4.2
4.2×10^{-3}	1.8
4.2×10^{-4}	0.3
4.2×10^{-5}	0.02
4.2×10^{-6}	0.002

The volatilization rate constants in the table above are based on an air temperature of 25°C and a pond depth of 1 meter. To reflect that the pond may be frozen for a portion of the year, the K_v values were multiplied by a seasonality factor of 9/12, or 0.75. The K_v values predicted using the Mills et al. table were also divided by 4.1 to adjust for the 4.1 meter average depth of a typical pond. The adjusted, annual averaged volatilization rate constants are shown in Table D-24. Henry's Law constants are presented in Table D-41 at the end of this appendix.

The adsorption equilibrium constant (K_d) for organics was estimating using the formula:

$$K_d = (F_{oc})(K_{oc})$$

Where:

F_{oc} = fraction of organic carbon in soil, 0.34 (Ecologistics 1987)

K_{oc} = soil adsorption coefficient based on organic carbon (mg/g)/(mg/cm^3)

K_{oc} values are summarized in Table D-41 at the end of this appendix. For metals, the K_d values shown in Table D-12 were used.

Pollutant concentrations in the hypothetical pond calculated using the approach outlined in this section are presented in Tables D-25 through D-27 for incinerator, evaporator, and fugitive emissions.

TABLE D-24

VOLATILIZATION RATE CONSTANTS

CONTAMINANT	Kv (1/d)
ACETONE	2.00E+01
ACRYLONITRILE	1.20E+02
BENZENE	3.14E+02
BENZO(A)ANTHRACENE	1.34E+00
BENZO(A)PYRENE	1.34E+00
BROMODICHLOROMETHANE	2.80E+02
BROMOFORM	2.80E+02
BUTADIENE,1,3-	3.20E+02
CARBON TETRACHLORIDE	3.14E+02
CHLOROBENZENE	3.14E+02
CHLOROFORM	3.14E+02
DICHLORODIFLUOROMETHANE	3.20E+02
DIOXIN,2,3,7,8-EQ(EPA)	2.00E+01
DIOXIN,2,3,7,8-EQ(MOE)	2.00E+01
ETHANOL	2.00E+01
ETHYLENE GLYCOL	3.20E+02
ETHYLENE OXIDE	1.20E+02
FORMALDEHYDE	1.20E+02
HEXACHLOROBENZENE	2.80E+02
HYDRAZINE	3.20E+02
METHYL ETHYL KETONE	1.20E+02
METHYLENE CHLORIDE	2.80E+02
NAPHTHALENE	2.80E+02
NITROPHENOL,o-	2.00E+01
PCB	2.80E+02
PETROLEUM DISTILLATES	3.20E+02
PHENOL	1.34E+00
PYRIDINE	3.20E+02
STYRENE	3.14E+02
TETRACHLOROETHYLENE	3.14E+02
TETRAETHYL LEAD	3.20E+02
TOLUENE	3.14E+02
TRICHLOROETHANE,1,1,1-	3.14E+02
TRICHLOROETHYLENE	3.14E+02
TRICHLOROTRIFLUOROETHANE	3.20E+02
XYLENE	3.14E+02

TABLE D-25

CONCENTRATION OF POLLUTANTS IN POND - INCINERATOR EMISSIONS

CONTAMINANT	POND CONC (mg/l)
ACETONE	6.53E-06
ACRYLONITRILE	7.00E-09
BENZENE	9.02E-08
BENZO(A)ANTHRACENE	3.31E-08
BENZO(A)PYRENE	4.65E-08
BROMODICHLOROMETHANE	3.43E-09
BROMOFORM	2.65E-10
BUTADIENE,1,3-	2.35E-10
CARBON TETRACHLORIDE	1.36E-08
CHLOROBENZENE	9.77E-10
CHLOROFORM	1.41E-07
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.07E-12
DIOXIN,2,3,7,8-EQ(MOE)	2.22E-11
ETHANOL	9.58E-06
ETHYLENE GLYCOL	7.83E-07
ETHYLENE OXIDE	5.33E-10
FORMALDEHYDE	1.89E-08
HEXACHLOROBENZENE	2.19E-10
HYDRAZINE	6.57E-13
METHYL ETHYL KETONE	3.05E-07
METHYLENE CHLORIDE	2.54E-07
NAPHTHALENE	1.05E-08
NITROPHENOL,o-	1.01E-07
PCB	2.34E-10
PETROLEUM DISTILLATES	6.91E-07
PHENOL	3.47E-07
PYRIDINE	1.32E-08
STYRENE	0.00E+00
TETRACHLOROETHYLENE	3.39E-07
TETRAETHYL LEAD	5.28E-11
TOLUENE	6.09E-07
TRICHLOROETHANE,1,1,1-	2.36E-07
TRICHLOROETHYLENE	3.01E-07
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	3.07E-07
ARSENIC	2.51E-07
BERYLLIUM	2.69E-09
CADMIUM	4.40E-06
CHROMIUM (VI)	1.09E-05
COPPER	2.53E-05
LEAD	1.36E-04
MERCURY	6.77E-06
NICKEL	2.96E-06
ZINC	3.83E-04

TABLE D-26

CONCENTRATION OF POLLUTANTS IN POND - EVAPORATOR EMISSIONS

CONTAMINANT	POND CONC mg/l
ACETONE	0.00E+00
ACRYLONITRILE	9.33E-08
BENZENE	6.92E-09
BENZO(A)ANTHRACENE	1.08E-09
BENZO(A)PYRENE	1.07E-08
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.71E-09
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	4.52E-07
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	4.33E-10
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	5.17E-07
NITROPHENOL,o-	0.00E+00
PCB	5.59E-12
PETROLEUM DISTILLATES	0.00E+00
PHENOL	7.50E-07
PYRIDINE	0.00E+00
STYRENE	6.66E-09
TETRACHLOROETHYLENE	2.33E-08
TETRAETHYL LEAD	0.00E+00
TOLUENE	1.23E-07
TRICHLOROETHANE,1,1,1-	4.62E-07
TRICHLOROETHYLENE	1.18E-08
TRICHLOROTRIFLUOROETHAN	4.52E-07
XYLENE	2.33E-08
ARSENIC	2.03E-10
BERYLLIUM	0.00E+00
CADMIUM	4.72E-12
CHROMIUM (VI)	2.46E-10
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	2.36E-10
ZINC	0.00E+00

TABLE D-27

CONCENTRATION OF POLLUTANTS IN POND - FUGITIVE EMISSIONS

CONTAMINANT	POND CONC mg/l
ACETONE	3.50E-04
ACRYLONITRILE	6.25E-07
BENZENE	9.43E-07
BENZO(A)ANTHRACENE	1.73E-16
BENZO(A)PYRENE	2.42E-16
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	3.60E-07
CARBON TETRACHLORIDE	4.35E-07
CHLOROBENZENE	0.00E+00
CHLOROFORM	1.08E-06
DICHLORODIFLUOROMETHANE	1.17E-06
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	2.42E-04
ETHYLENE GLYCOL	9.76E-07
ETHYLENE OXIDE	1.10E-08
FORMALDEHYDE	3.16E-05
HEXACHLOROBENZENE	2.92E-15
HYDRAZINE	1.28E-11
METHYL ETHYL KETONE	9.52E-06
METHYLENE CHLORIDE	2.45E-05
NAPHTHALENE	3.64E-11
NITROPHENOL,o-	0.00E+00
PCB	2.34E-11
PETROLEUM DISTILLATES	1.09E-05
PHENOL	5.44E-06
PYRIDINE	2.38E-07
STYRENE	9.03E-09
TETRACHLOROETHYLENE	6.02E-06
TETRAETHYL LEAD	8.02E-10
TOLUENE	1.62E-05
TRICHLOROETHANE,1,1,1-	7.04E-06
TRICHLOROETHYLENE	8.12E-06
TRICHLOROTRIFLUOROETHANE	1.82E-07
XYLENE	4.95E-06

D.5 ESTIMATION OF CHEMICAL CONCENTRATIONS IN PLANTS

Human exposure to chemicals released from the OWMC facility may occur through a variety of food pathways, including ingestion of fruits, vegetables, meat, milk, and eggs. To predict the risk associated with consumption of these foods, the concentration of chemicals in plants ingested by humans was estimated as part of the Phase 4B assessment. Specifically, consumption of the following types of produce was evaluated:

- Leafy vegetables, which present a leafy, edible surface exposed to the atmosphere. Examples include lettuce and broccoli.
- Exposed produce, which also present edible surfaces exposed to the atmosphere, but which are not leafy in nature. Examples include tomatoes and apples.
- Unexposed produce, which do not have edible surfaces exposed to the atmosphere. Examples include potatoes and corn.

Similarly, the contaminant concentrations in crops that may be used as feed for cattle, swine, and poultry were estimated, since these animal products were also considered as human food sources in the Phase 4B risk analysis (see D.6). It was assumed that the crops consumed by livestock would be hay, mixed grains, and/or corn silage.

In the following sections, the steps used to estimate concentrations in both human food and livestock feed are described. Consideration is given to both the mechanisms for chemical accumulation in plants and for removal mechanisms.

D.5.1 MODEL FOR ESTIMATING CONCENTRATIONS IN PLANTS

The model for chemical accumulation in plants considers both deposition onto leaf surfaces by dry and wet mechanisms and uptake from soil through the root system. A third mechanism of uptake by plants, foliar absorption of vapor phase organics, was also considered. However, based on results generated using a model developed by O'Dell et al. (1977), it was determined that this mechanism would not be significant compared to direct deposition and root uptake as modeled in the risk assessment. It should be noted that the relative insignificance of foliar uptake for certain chemicals may be the result of conservative assumptions made in estimating the contribution associated with direct deposition and/or root uptake. It is possible that, in actuality, foliar uptake may be the most important uptake mechanism for some chemicals.

The following equation was used to estimate the extent of chemical accumulation in plants (Travis et al. 1983):

$$C_p = \frac{dr[1 - e^{-Kt}]}{Y \ K} + BC_s$$

Where:

C_p = concentration of chemical in edible plant material, µg/kg

d = total chemical deposition rate, $\mu g/m^2$-s

K = total rate constant for plant leaf degradation processes, s^{-1}

t = length of the growing season, s

Y = crop yield, kg/m^2

r = interception fraction of plant, dimensionless

C_s = concentration of chemical in root zone soil, µg/kg

B = soil/plant bioaccumulation factor, dimensionless

The equation uses deposition rates and soil concentrations calculated in Sections D.3.1 and D.3.2, respectively. Examination of the equation indicates that it also contains plant-specific parameters related to chemical accumulation (crop yield, interception fraction, and soil/plant bioaccumulation factor) as well as a rate constant for plant leaf degradation processes. The estimation of these parameters is described separately in Sections D.5.2 and D.5.3, respectively.

Inputs for crop-specific data used in the analysis are shown in Table D-28. In the assessment, a growing season of five months was assumed.

TABLE D-28

INPUTS FOR CROP-SPECIFIC DATA

	Crop Yield[1] (y) kg/m^2 wet weight	Fraction[2] Water, (f)	Crop Yield[3] (y) kg/m^2 dry weight	Interception Fraction[4] (r)	Human Intake[5] (I) kg/d
Leafy Vegetables					
Cabbage	4.100	0.924	0.312	0.29	0.018
Lettuce	2.157	0.948	0.112	0.17	0.026
Broccoli	0.885	0.899	0.089	0.07	0.006
Cauliflower	2.098	0.917	0.174	0.16	0.008
Exposed Produce					
Asparagus	0.168	0.841	0.027	0.01	0.001
Tomatoes	4.246	0.941	0.250	0.13	0.074
Pears & Plums	1.604	0.827	0.277	0.05	0.007
Cherries	0.585	0.83	0.099	0.02	0.010
Peaches	0.778	0.869	0.102	0.02	0.008
Apples	1.371	0.841	0.218	0.04	0.054
Strawberries	0.851	0.899	0.086	0.03	0.006
Grapes	0.631	0.819	0.114	0.02	0.002
Beans	1.166	0.889	0.129	0.04	0.006
Cucumbers	2.006	0.961	0.078	0.06	0.007
Protected Produce					
Beets	1.238	0.836	0.203	0	0.002
Carrots	6.458	0.882	0.762	0	0.027
Corn	1.123	0.739	0.293	0	0.027
Onions	4.246	0.875	0.531	0	0.018
Peas	0.357	0.743	0.092	0	0.007
Rutabagas	4.394	0.778	0.975	0	0.007
Potatoes	2.345	0.778	0.521	0	0.182
Fodder					
Hay	0.815	0.12	0.718	0.87	--
Corn	3.361	0.65	1.176	0.60	--
Mixed Grains	0.296	0.12	0.260	0.53	--

---

[1] Ecologistics 1984a.
[2] Ecologistics 1984a.
[3] Calculated as Crop Yield, wet weight, multiplied by (1-f).
[4] Calculated using the method of Baes et al. 1984.
[5] Statistics 1985.

D.5.2 PARAMETERS FOR CHEMICAL ACCUMULATION

D.5.2.1 Crop Yield

Crop yield is an important factor in predicting the chemical concentrations in a plant. High crop yield has the effect of "diluting" the pollutant concentration in the plant by lowering the surface deposition factor. Yield data used in the Phase 4B risk assessment were obtained from Ecologistics (1984a) for food and feed crops grown in the region of the proposed OWMC facility. These values are shown in Table D-28. An overall weighted-average crop yield value for each food crop category was determined based on the average daily consumption of the individual foods contained in each category as follows:

$$Y = \sum_i (Y_i)(I_i/I_t)$$

Where:

Y = weighted-average crop yield (dry weight), kg/m^2

Y_i = crop yield (dry weight) from crop i, kg/m^2

I_i = human intake for crop i, kg/d

I_t = total human intake for crops in the category, kg/d

Each type of fodder (hay, corn, and mixed grains) was treated separately and so that it was not necessary to calculate weighted-average crop yields. The crop yields used in the risk assessment are summarized in Table D-29.

TABLE D-29

CROP-SPECIFIC INFORMATION

Food Crop	Crop Yield, Y kg/m^2 dry weight	Fraction Water
Leafy vegetables	0.180	0.931
Exposed produce	0.207	0.892
Protected produce	0.521	0.791
Hay	0.718	NA
Corn	1.176	NA
Mixed grains	0.260	NA

NA - Not Applicable

Source: Ecologistics (1984a)

D.5.2.2 Interception Fraction

The interception fraction is a measure of the portion of a plant that is available for deposition. Thus, the interception fraction is used with the ground level deposition rate to determine the fraction of a chemical intercepted by the edible surface of a plant. Since data on interception fractions for specific vegetables and field crops were unavailable, these values were calculated using a theoretical model developed by Baes et al. (1984). This model, which is based on a theoretical analysis of field crop geometries, can be used to predict interception fractions for each type of plant considered (Baes et al. 1984):

$$r_{lv} = 1.0 - \exp(-0.0846\, Y_{lv})$$

$$r_e = 1.0 - \exp(-0.0324\, Y_e)$$

$$r_s = 1.0 - \exp(-0.769\, Y_s)$$

$$r_h = 1.0 - \exp(-2.88\, Y_h)$$

$$r_{pg} = 1.0 - \exp(-2.88\, Y_{pg})$$

Where:

r = interception fraction
Y = yield of the plant, kg/m^2
lv = leafy vegetables
e = exposed produce
s = silage
h = hay
pg = pasture grasses

It was assumed that an interception fraction for mixed grains could be approximated to that used for pasture grass and hay.

The calculation of interception fraction values requires that a distinction be made between the air-dry weight and the fresh weight yields. Feed crop productivities (hay, corn silage, and mixed grains) are considered to be on an air-dry weight basis and food crops (vegetables and produce) productivities are on a fresh weight basis (Baes et al. 1984).

As with crop yield, interception fractions for each individual crop within each crop category were calculated, and a weighted average based on daily intake was used to represent the entire crop category. The data used are presented in Table D-29. The weighted-average interception fractions calculated in this manner are presented in Table D-30.

D.5.2.3 Soil/Plant Bioaccumulation

The soil/plant bioaccumulation factor can be defined as the ratio of the concentration of a chemical in the plant to the concentration of the chemical in the soil. These factors apply only to equilibrium or steady state conditions. Bioaccumulation will occur to varying degrees in the non-vegetative (reproductive) and vegetative portions of plants, so that different soil/plant bioaccumulation factors

TABLE D-30

WEIGHTED-AVERAGE INTERCEPTION FRACTIONS

Food Crop	Weighted-average Interception Fraction, (r) dimensionless
Leafy vegetables	0.196
Exposed produce	0.077
Protected produce	0
Hay	0.87
Corn	0.60
Mixed grains	0.53

correspond to non-vegetative (reproductive) and vegetative portions of plants (Baes et al. 1984). Vegetative growth includes leaves and stems while non-vegetative growth includes fruits, seeds, and tubers. The crops considered in the risk assessment were categorized according to the nature of the edible portion as follows:

Crop	Crop Type
Leafy vegetables	vegetative
Exposed produce	reproductive
Protected produce	reproductive
Mixed grains	reproductive
Hay	vegetative
Corn silage	vegetative

Bioaccumulation factors for organic chemicals in vegetative growth have been found to be positively correlated with octanol-water partition coefficients. A regression equation based on octanol-water partition coefficients has been derived by Travis et al. (1987) using measured values of the accumulation of 29 organic chemicals in vegetation:

$$\log B_v = 1.2017 - 0.5534 (\log Kow)$$

Where:

B_v = soil/plant bioaccumulation factor for vegetative portions, dimensionless

K_{ow} = octanol-water partition coefficient, dimensionless (presented in Table D-41 at the end of this appendix)

This regression equation was used in the risk assessment to estimate the vegetative bioaccumulation factor (B_v) for all organic chemicals except polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF), polychlorinated biphenyls (PCBs), hexachlorobenzene, benzo(a)pyrene, and benzo(a)-anthracene. The B_v values for these chemicals were based on actual measurements, and are discussed below.

PCDD/PCDF. The B_v value for PCDD/PCDF was based on a study conducted by Helling et al. (1973), in which two forms of PCDD, dichlorodioxin (DCDD) and tetrachlorodioxin (TCDD) were applied to a soil with low adsorptive capacity. The concentrations of DCDD and TCDD in the soil were 100 ppb and 60 ppb, respectively. Measured concentrations in mature oats and soybean tops planted in the soil ranged from <1 ppb to 20 ppb for DCDD, and <1 ppb for TCDD. Chemicals that absorb strongly to soil are less available for plant uptake than those that are weakly adsorbed. Since highly chlorinated dioxins are more likely to adsorb to soil than less chlorinated ones, DCDD may be more readily taken up by the root system of a plant than TCDD. The PCDD and PCDF of greatest concern, however, are substituted with at lease four chlorine atoms. The data corresponding to TCDD uptake therefore was used to estimate the biotransfer factor for PCDD/PCDF:

$$B_V = \frac{1 \text{ ppb}}{60 \text{ ppb}} = 0.017$$

PCBs. The B_v value for PCBs was based on an experiment conducted by Iwata and Gunther (1976). According to Iwata and

Gunther, carrot foliage was measured to contain between 1 percent and 6 percent (in ppm) of the PCB residues in soil. The maximum B_v value is therefore 0.06. It should be noted that the authors conclude that "the foliar residues probably resulted from a combination of root sorption with subsequent translocation and residues from soil dust adhering to the foliage".

Hexachlorobenzene. Dejonckheere et al. (1975) reported hexachlorobenzene concentrations in contaminated soil and lettuce grown in that soil. The ratios of hexachlorobenzene concentration in lettuce to the concentration in soil ranged from 0.12 to 1.6. Based on these data, a B_v value of 1.6 was conservatively assumed.

Benzo(a)pyrene and Benzo(a)anthracene. In a review of the environmental fate and transport of polycyclic aromatic hydrocarbons, Edwards (1983) reports that, for benzo(a)pyrene, concentrations in vegetation are between 3 and 10 000 times lower than corresponding concentrations in soil. These results may reflect not only uptake through the root system, but deposition of contaminated soil dust as well. In his review of the data, Travis et al. (1987) concluded that a B_v value of 0.06 is appropriate for the purposes of estimating root uptake of benzo(a)pyrene. This value has therefore been applied in the risk assessment. The same value was also used for benzo(a)anthracene, which is similar to benzo(a)pyrene, both in terms of structure and physical/chemical properties.

To estimate the accumulation of organic chemicals in the non-vegetative portion of plants, it was assumed that accumulation is approximately 10 times lower than in the vegetative portion. This assumption is based on work conducted by Baes et al. (1984). Therefore:

$$B_r = 0.1B_v$$

Where:

B_v = soil/plant bioaccumulation factor for vegetative portions, dimensionless

B_r = soil/plant bioaccumulation factor for nonvegetative (reproductive) portions, dimensionless

Values for B_v and B_r for the organic chemicals of concern are shown in Table D-31. It should be noted that there may be considerable uncertainty associated with the biotransfer factors based on the generalized correlations rather than actual measurements.

For the accumulation of metals, Baes et al. (1984) have measured the uptake of radionuclides in soil by the roots of a variety of plants. The soil/plant bioaccumulation factors estimated by Baes et al. (1984) for vegetative and non-vegetative crops have been used for metals in this analysis. These values are presented in Table D-31.

TABLE D-31

SOIL/PLANT BIOACCUMULATION FACTOR VALUES

FOOD PATHWAYS CHEMICAL CONSTITUENT	VEGETATIVE PLANT-SOIL PARTITION COEFF. (Bv)	REPRODUCTIVE PLANT-SOIL PARTITION COEFF. (Br)
ACETONE	21.61	2.161
ACRYLONITRILE	11.57	1.157
BENZENE	1.06	0.106
BENZO(A)ANTHRACENE	0.06	0.006
BENZO(A)PYRENE	0.06	0.006
BROMODICHLOROMETHANE	1.10	0.110
BROMOFORM	0.77	0.077
BUTADIENE,1,3-	1.26	0.126
CARBON TETRACHLORIDE	0.43	0.043
CHLOROBENZENE	0.43	0.043
CHLOROFORM	1.30	0.130
DICHLORODIFLUOROMETHANE	1.13	0.113
DIOXIN,2,3,7,8-EQ(EPA)	0.02	0.002
DIOXIN,2,3,7,8-EQ(MOE)	0.02	0.002
ETHANOL	23.93	2.393
ETHYLENE GLYCOL	185.90	18.590
ETHYLENE OXIDE	23.36	2.336
FORMALDEHYDE	36.43	3.643
HEXACHLOROBENZENE	1.60	0.160
HYDRAZINE	91.12	9.112
METHYL ETHYL KETONE	11.43	1.143
METHYLENE CHLORIDE	3.20	0.320
NAPHTHALENE	0.24	0.024
NITROPHENOL,o-	1.71	0.171
PCB	0.06	0.006
PETROLEUM DISTILLATES	6.78	0.678
PHENOL	2.48	0.248
PYRIDINE	7.05	0.705
STYRENE	0.37	0.037
TETRACHLOROETHYLENE	0.58	0.058
TETRAETHYL LEAD	0.05	0.005
TOLUENE	0.52	0.052
TRICHLOROETHANE,1,1,1-	0.65	0.065
TRICHLOROETHYLENE	0.73	0.073
TRICHLOROTRIFLUOROETHANE	0.21	0.021
XYLENE	0.32	0.032
ARSENIC	0.04	0.006
BERYLLIUM	0.01	0.002
CADMIUM	0.55	0.150
CHROMIUM (VI)	0.0075	0.005
COPPER	0.4	0.250
LEAD	0.045	0.009
MERCURY	0.9	0.200
NICKEL	0.06	0.060
ZINC	1.5	0.900

D.5.3 REMOVAL MECHANISMS

Chemicals on a leaf surface may be lost as a result of several removal mechanisms, including photolysis, weathering due to washoff by precipitation, and volatilization. These processes can decrease the amount of chemical present on a leaf surface. In the Phase 4B risk assessment, consideration was given to the effects of weathering and volatilization only as removal mechanisms. These processes are described in the following subsections, and an estimate of the total rate constant for plant leaf degradation is developed.

D.5.3.1 Weathering

The rate constant for weathering (K_w) describes the loss of a deposited chemical due to wash off by precipitation. The weathering rate constant is defined by:

$$K_w = \frac{0.693}{t_{1/2}}$$

Where:

K_w = weathering rate constant, s^{-1}

$t_{1/2}$ = half-life of chemical on the leaf due to washoff

A weathering half-life of 15 days was assumed (Willis et al. 1980) for all the chemicals modeled in the risk assessment. This half-life was based on leaf wash-off of the pesticide endrin, a highly hydrophobic chemical that would be expected to adsorb strongly to soil or leaf surfaces. The weathering half-life of 15 days is comparable to the 14 day half-life used by Baes et al. (1984) for the wash-off of metals from a leaf surface. The weathering rate constant corresponding to a 15 day half-life is 5.3×10^{-7} s^{-1}.

D.5.3.2 Volatilization

Some chemicals present on a leaf surface may be released as vapors to the atmosphere. This process, known as volatilization, may occur after dry deposition of the chemical onto the leaf surface, or as leaf surface moisture containing the chemical evaporates.

In the risk assessment, volatilization was considered only for organic chemicals since most metals have extremely low vapor pressures and thus would not be expected to volatilize to any appreciable extent. Although it was recognized that some metals, such as mercury, may volatilize, data with which to predict such losses were unavailable. Furthermore, volatilization of PCDD/PCDF from the plant surface was not considered since it may be bound tightly to fly ash emitted from the incinerator.

A model describing the volatilization of organics from plant surfaces is not currently available. Therefore, an empirically derived soil surface volatilization model was used to predict the rate of release for organics from a leaf surface (Lyman et al., 1982). The model incorporates certain chemical properties such as vapor pressure, water solubility and organic carbon adsorption which are also significant in predicting the volatilization of chemicals from a leaf surface as well as from soil. Assuming that a leaf surface contains 100% organic carbon, the volatilization rate constant can be estimated using the following formula:

$$K_V = 509 \frac{P}{K_{OC}\, S}$$

Where:

K_V = volatilization rate constant, s^{-1}

P = vapor pressure of the chemical, mm Hg

S = water solubility of the chemical, mg/l

K_{OC} = soil (organic carbon) adsorption coefficient, (g/g)/(g/ml)

Data with respect to vapor pressure, solubility, and soil adsorption coefficient are shown in Table D-41 at the end of the appendix.

D.5.3.3 Total Degradation Rate Constant

Based on the estimation of losses due to weathering and volatilization, a total rate constant for plant leaf degradation processes was derived as follows:

$$K = K_V + K_W$$

Where:

K = total rate constant for plant surface degradation, s^{-1}

K_V = rate constant for volatilization from the leaf surface, s^{-1}

K_W = rate constant for wash off due to precipitation, s^{-1}

As previously discussed, it has been assumed that metals and PCDD/PCDF deposited onto leaf surfaces would not be lost through volatilization. Thus, the total rate constant for plant leaf degradation processes for metals and PCDD/PCDF equals the rate constant for wash off due to precipitation. The total rate constants for the organic chemicals of concern are presented in Table D-32.

D.5.4 ESTIMATED CHEMICAL CONCENTRATIONS IN PLANTS

Chemical concentrations in the various types of crops were calculated on a dry weight basis by incorporating the total rate constant for plant surface degradation (Section D.5.3) and the plant-specific parameters for chemical accumulation (Section D.5.2) into the model for accumulation in plants (Section D.5.2). The estimated concentrations in food crops were converted to a fresh weight equivalent basis by dividing by the dry weight fraction (derived from Table D-29). Chemical concentrations in feed crops are presented on a dry weight

TABLE D-32

PLANT SURFACE DEGRADATION RATE CONSTANTS

CHEMICAL CONSTITUENT	PLANT VOLATIZ. RATE CONSTANT Kpv (1/s)	TOTAL PLANT SURFACE DEG. RATE Ka (1/s)
ACETONE	1.13E-02	1.13E-02
ACRYLONITRILE	7.58E-01	7.58E-01
BENZENE	4.16E-01	4.16E-01
BENZO(A)ANTHRACENE	2.23E-09	5.32E-07
BENZO(A)PYRENE	1.36E-10	5.30E-07
BROMODICHLOROMETHANE	9.27E-02	9.27E-02
BROMOFORM	7.23E-03	7.23E-03
BUTADIENE,1,3-	1.25E+01	1.25E+01
CARBON TETRACHLORIDE	1.13E-01	1.13E-01
CHLOROBENZENE	3.73E-02	3.73E-02
CHLOROFORM	2.41E-01	2.41E-01
DICHLORODIFLUOROMETHANE	1.33E+02	1.33E+02
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	5.30E-07
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	5.30E-07
ETHANOL	1.86E-03	1.87E-03
ETHYLENE GLYCOL	4.08E-09	5.34E-07
ETHYLENE OXIDE	1.62E-02	1.62E-02
FORMALDEHYDE	3.07E-01	3.07E-01
HEXACHLOROBENZENE	1.19E-06	1.72E-06
HYDRAZINE	1.89E-03	1.89E-03
METHYL ETHYL KETONE	1.44E+01	1.44E+01
METHYLENE CHLORIDE	1.50E+00	1.50E+00
NAPHTHALENE	1.70E-03	1.70E-03
NITROPHENOL,o-	1.36E-03	1.36E-03
PCB	2.39E-06	2.92E-06
PETROLEUM DISTILLATES	1.36E+00	1.36E+00
PHENOL	1.93E-04	1.93E-04
PYRIDINE	1.32E-03	1.32E-03
STYRENE	3.85E-02	3.85E-02
TETRACHLOROETHYLENE	1.31E-01	1.31E-01
TETRAETHYL LEAD	3.42E-04	3.42E-04
TOLUENE	9.30E-02	9.30E-02
TRICHLOROETHANE,1,1,1-	7.61E-02	7.61E-02
TRICHLOROETHYLENE	2.71E-01	2.71E-01
TRICHLOROTRIFLUOROETHANE	1.87E+00	1.87E+00
XYLENE	6.43E-02	6.43E-02
ARSENIC		5.30E-07
BERYLLIUM		5.30E-07
CADMIUM		5.30E-07
CHROMIUM (VI)		5.30E-07
COPPER		5.30E-07
LEAD		5.30E-07
MERCURY		5.30E-07
NICKEL		5.30E-07
ZINC		5.30E-07

basis. The resulting estimates of chemical concentrations in plants are shown in Tables D-33, D-34, and D-35 for incinerator, evaporator and fugitive emissions, respectively.

D.6 ESTIMATION OF CHEMICAL CONCENTRATIONS IN MEAT, MILK, AND EGGS

To predict risks associated with the consumption of meat, milk, and eggs, the concentration of chemicals in these foods was estimated as part of the Phase 4B risk assessment. Consistent with the known activities in the vicinity of the preferred site, both poultry and cattle have been considered in the estimation of chemical concentrations in meat.

In the following section, the steps used to estimate chemical concentrations in these food products are described.

D.6.1 MODEL FOR ESTIMATING CHEMICAL CONCENTRATIONS

The calculation of chemical concentrations in meat, eggs, and milk was based on a model developed by Holton et al. (1984) for ingestion of contaminated feed. For the purposes of the Phase 4B risk assessment, the model has been extended to include

TABLE D-33

CONCENTRATION OF CHEMICALS IN PLANTS - INCINERATOR EMISSIONS

CHEMICAL CONSTITUENT	LEAFY VEGETABLE CONC (ug/kg)	EXPOSED PRODUCE CONC (ug/kg)	PROTECTED PRODUCE CONC (ug/kg)	CORN SILAGE CONC (ug/kg)	HAY CONC (ug/kg)	GRAINS CONC (ug/kg)
ACETONE	6.30E-01	9.87E-02	1.91E-01	9.13E+00	9.13E+00	9.15E-01
ACRYLONITRILE	1.83E-03	2.86E-04	5.54E-04	2.65E-02	2.65E-02	2.65E-03
BENZENE	2.56E-03	4.01E-04	7.75E-04	3.71E-02	3.71E-02	3.72E-03
BENZO(A)ANTHRACENE	7.26E-04	3.36E-04	4.19E-05	5.98E-03	1.15E-02	1.61E-02
BENZO(A)PYRENE	3.02E-04	1.36E-04	2.00E-05	2.55E-03	4.75E-03	6.49E-03
BROMODICHLOROMETHANE	1.86E-04	2.91E-05	5.63E-05	2.70E-03	2.70E-03	2.71E-04
BROMOFORM	4.38E-05	6.87E-06	1.33E-05	6.34E-04	6.35E-04	6.48E-05
BUTADIENE,1,3-	1.37E-06	2.15E-07	4.15E-07	1.99E-05	1.99E-05	1.99E-06
CARBON TETRACHLORIDE	2.03E-04	3.19E-05	6.15E-05	2.94E-03	2.95E-03	2.99E-04
CHLOROBENZENE	7.78E-05	1.22E-05	2.36E-05	1.13E-03	1.13E-03	1.14E-04
CHLOROFORM	5.38E-03	8.43E-04	1.63E-03	7.80E-02	7.80E-02	7.82E-03
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.13E-07	5.68E-08	2.83E-09	8.36E-07	1.80E-06	2.82E-06
DIOXIN,2,3,7,8-EQ(MOE)	2.34E-06	1.18E-06	5.89E-08	1.74E-05	3.74E-05	5.86E-05
ETHANOL	1.04E+00	1.63E-01	3.15E-01	1.51E+01	1.51E+01	1.52E+00
ETHYLENE GLYCOL	1.09E+01	2.57E+00	2.59E+00	1.40E+02	1.61E+02	7.48E+01
ETHYLENE OXIDE	3.30E-04	5.16E-05	9.98E-05	4.78E-03	4.78E-03	4.78E-04
FORMALDEHYDE	9.52E-03	1.49E-03	2.88E-03	1.38E-01	1.38E-01	1.38E-02
HEXACHLOROBENZENE	2.84E-03	4.84E-04	8.29E-04	4.04E-02	4.13E-02	6.78E-03
HYDRAZINE	3.63E-06	5.69E-07	1.10E-06	5.27E-05	5.27E-05	5.28E-06
METHYL ETHYL KETONE	9.56E-02	1.50E-02	2.89E-02	1.38E+00	1.38E+00	1.38E-01
METHYLENE CHLORIDE	1.03E-02	1.61E-03	3.12E-03	1.49E-01	1.49E-01	1.49E-02
NAPHTHALENE	2.16E-03	3.41E-04	6.50E-04	3.12E-02	3.13E-02	3.34E-03
NITROPHENOL,o-	2.52E-03	3.97E-04	7.61E-04	3.65E-02	3.65E-02	3.85E-03
PCB	2.13E-04	6.52E-05	3.88E-05	2.43E-03	3.22E-03	2.48E-03
PETROLEUM DISTILLATES	3.38E-02	5.29E-03	1.02E-02	4.90E-01	4.90E-01	4.90E-02
PHENOL	1.03E-03	1.68E-04	3.05E-04	1.47E-02	1.49E-02	1.97E-03
PYRIDINE	9.34E-03	1.47E-03	2.82E-03	1.35E-01	1.35E-01	1.39E-02
STYRENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	9.61E-03	1.51E-03	2.91E-03	1.39E-01	1.39E-01	1.40E-02
TETRAETHYL LEAD	3.69E-06	6.69E-07	1.05E-06	5.16E-05	5.39E-05	1.15E-05
TOLUENE	2.88E-02	4.51E-03	8.72E-03	4.17E-01	4.17E-01	4.20E-02
TRICHLOROETHANE,1,1,1-	3.53E-03	5.54E-04	1.07E-03	5.11E-02	5.11E-02	5.24E-03
TRICHLOROETHYLENE	6.69E-03	1.05E-03	2.03E-03	9.70E-02	9.70E-02	9.75E-03
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
XYLENE	1.16E-02	1.81E-03	3.50E-03	1.68E-01	1.68E-01	1.70E-02
ARSENIC	4.49E-03	2.18E-03	3.33E-04	3.61E-02	7.11E-02	1.04E-01
BERYLLIUM	4.22E-05	2.19E-05	1.03E-06	3.03E-04	6.76E-04	1.09E-03
CADMIUM	1.84E-01	8.56E-02	9.72E-02	2.15E+00	2.77E+00	2.26E+00
CHROMIUM (VI)	1.68E-01	9.29E-02	1.27E-02	1.19E+00	2.70E+00	4.44E+00
COPPER	7.04E-01	5.21E-01	6.15E-01	7.28E+00	1.08E+01	1.32E+01
LEAD	2.54E+00	1.24E+00	3.18E-01	2.12E+01	4.00E+01	5.62E+01
MERCURY	1.64E-01	7.60E-02	4.18E-02	1.58E+00	2.53E+00	2.96E+00
NICKEL	5.65E-02	4.27E-02	3.66E-02	4.76E-01	8.90E-01	1.38E+00
ZINC	2.62E+01	2.23E+01	3.71E+01	3.35E+02	3.89E+02	3.34E+02

TABLE D-34

CONCENTRATION OF CHEMICALS IN PLANTS - EVAPORATOR EMISSIONS

CHEMICAL CONSTITUENT	LEAFY PLANT CONC (ug/kg)	EXPOSED PRODUCE CONC (ug/kg)	PROTECTED PRODUCE CONC (ug/kg)	CORN SILAGE CONC (ug/kg)	HAY CONC (ug/kg)	GRAINS CONC (ug/kg)
ACETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ACRYLONITRILE	2.44E-02	3.82E-03	7.39E-03	3.53E-01	3.53E-01	3.53E-02
BENZENE	1.96E-04	3.07E-05	5.94E-05	2.84E-03	2.84E-03	2.85E-04
BENZO(A)ANTHRACENE	2.37E-05	1.10E-05	1.37E-06	1.96E-04	3.74E-04	5.28E-04
BENZO(A)PYRENE	6.94E-05	3.14E-05	4.60E-06	5.87E-04	1.09E-03	1.49E-03
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	9.98E-06	1.56E-06	3.02E-06	1.45E-04	1.45E-04	1.45E-05
CARBON TETRACHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	1.74E-04	2.72E-05	5.27E-05	2.52E-03	2.52E-03	2.52E-04
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
FORMALDEHYDE	2.18E-04	3.42E-05	6.61E-05	3.16E-03	3.16E-03	3.16E-04
HEXACHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NAPHTHALENE	1.06E-01	1.68E-02	3.21E-02	1.54E+00	1.54E+00	1.65E-01
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	5.07E-06	1.56E-06	9.26E-07	5.80E-05	7.67E-05	5.91E-05
PETROLEUM DISTILLATES	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PHENOL	2.22E-03	3.63E-04	6.60E-04	3.18E-02	3.22E-02	4.26E-03
PYRIDINE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
STYRENE	3.54E-04	5.54E-05	1.07E-04	5.12E-03	5.12E-03	5.19E-04
TETRACHLOROETHYLENE	6.61E-04	1.04E-04	2.00E-04	9.57E-03	9.58E-03	9.65E-04
TETRAETHYL LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TOLUENE	5.82E-03	9.12E-04	1.76E-03	8.44E-02	8.44E-02	8.49E-03
TRICHLOROETHANE,1,1,1-	6.90E-03	1.08E-03	2.09E-03	1.00E-01	1.00E-01	1.02E-02
TRICHLOROETHYLENE	2.62E-04	4.10E-05	7.94E-05	3.80E-03	3.80E-03	3.81E-04
TRICHLOROTRIFLUOROETHAN	3.26E-04	5.12E-05	9.87E-05	4.73E-03	4.73E-03	4.83E-04
XYLENE	8.79E-04	1.38E-04	2.66E-04	1.27E-02	1.27E-02	1.29E-03
ARSENIC	3.63E-06	1.76E-06	2.69E-07	2.91E-05	5.74E-05	8.37E-05
BERYLLIUM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CADMIUM	1.97E-07	9.18E-08	1.04E-07	2.31E-06	2.97E-06	2.42E-06
CHROMIUM (VI)	3.81E-06	2.10E-06	2.88E-07	2.70E-05	6.10E-05	1.00E-04
COPPER	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NICKEL	4.51E-06	3.40E-06	2.92E-06	3.80E-05	7.10E-05	1.10E-04
ZINC	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00

TABLE D-35

CONCENTRATION OF CHEMICALS IN PLANTS - FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	LEAFY PLANT CONC (ug/kg)	EXPOSED PRODUCE CONC (ug/kg)	PROTECTED PRODUCE CONC (ug/kg)	CORN SILAGE CONC (ug/kg)	HAY CONC (ug/kg)	GRAINS CONC (ug/kg)
ACETONE	3.38E+01	5.30E+00	1.02E+01	4.90E+02	4.90E+02	4.91E+01
ACRYLONITRILE	1.63E-01	2.56E-02	4.95E-02	2.37E+00	2.37E+00	2.37E-01
BENZENE	2.67E-02	4.18E-03	8.09E-03	3.87E-01	3.87E-01	3.88E-02
BENZO(A)ANTHRACENE	3.79E-12	1.75E-12	2.19E-13	3.12E-11	5.98E-11	8.42E-11
BENZO(A)PYRENE	1.57E-12	7.10E-13	1.04E-13	1.33E-11	2.47E-11	3.38E-11
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.10E-03	3.29E-04	6.36E-04	3.04E-02	3.04E-02	3.04E-03
CARBON TETRACHLORIDE	6.48E-03	1.02E-03	1.96E-03	9.39E-02	9.39E-02	9.54E-03
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	4.11E-02	6.43E-03	1.24E-02	5.95E-01	5.95E-01	5.97E-02
DICHLORODIFLUOROMETHANE	4.48E-04	7.02E-05	1.36E-04	6.50E-03	6.50E-03	6.50E-04
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	2.63E+01	4.12E+00	7.96E+00	3.81E+02	3.81E+02	3.84E+01
ETHYLENE GLYCOL	1.35E+01	3.20E+00	3.23E+00	1.74E+02	2.01E+02	9.32E+01
ETHYLENE OXIDE	6.83E-03	1.07E-03	2.07E-03	9.90E-02	9.90E-02	9.91E-03
FORMALDEHYDE	1.60E+01	2.50E+00	4.83E+00	2.31E+02	2.31E+02	2.31E+01
HEXACHLOROBENZENE	3.78E-08	6.44E-09	1.10E-08	5.37E-07	5.50E-07	9.02E-08
HYDRAZINE	7.07E-05	1.11E-05	2.14E-05	1.02E-03	1.02E-03	1.03E-04
METHYL ETHYL KETONE	2.99E+00	4.68E-01	9.05E-01	4.33E+01	4.33E+01	4.33E+00
METHYLENE CHLORIDE	9.93E-01	1.56E-01	3.01E-01	1.44E+01	1.44E+01	1.44E+00
NAPHTHALENE	7.51E-06	1.19E-06	2.27E-06	1.09E-04	1.09E-04	1.16E-05
NITROPHENOL,O-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	2.13E-05	6.53E-06	3.89E-06	2.43E-04	3.22E-04	2.48E-04
PETROLEUM DISTILLATES	5.34E-01	8.36E-02	1.62E-01	7.74E+00	7.74E+00	7.75E-01
PHENOL	1.61E-02	2.63E-03	4.79E-03	2.31E-01	2.34E-01	3.09E-02
PYRIDINE	1.68E-01	2.65E-02	5.09E-02	2.44E+00	2.44E+00	2.51E-01
STYRENE	4.80E-04	7.52E-05	1.45E-04	6.95E-03	6.95E-03	7.04E-04
TETRACHLOROETHYLENE	1.71E-01	2.67E-02	5.16E-02	2.47E+00	2.47E+00	2.49E-01
TETRAETHYL LEAD	5.61E-05	1.02E-05	1.59E-05	7.84E-04	8.18E-04	1.75E-04
TOLUENE	7.65E-01	1.20E-01	2.32E-01	1.11E+01	1.11E+01	1.12E+00
TRICHLOROETHANE,1,1,1-	1.05E-01	1.65E-02	3.18E-02	1.52E+00	1.53E+00	1.56E-01
TRICHLOROETHYLENE	1.80E-01	2.82E-02	5.44E-02	2.61E+00	2.61E+00	2.63E-01
TRICHLOROTRIFLUOROETHANE	1.31E-04	2.06E-05	3.98E-05	1.90E-03	1.91E-03	1.94E-04
XYLENE	1.87E-01	2.92E-02	5.65E-02	2.70E+00	2.70E+00	2.73E-01

doses received from breathing, drinking water, and consumption of contaminated soil. Incorporating these aspects, the chemical concentration in meat, milk, or eggs is given by:

$$C_M = F(f_F Q_F C_F + 1000\ f_W Q_W C_W + 1000\ f_A Q_A C_A + f_S Q_S C_S)$$

Where:

C_M = estimated concentration of the contaminant in meat, milk, or eggs, μg/kg

F = biotransfer factor in meat, milk, or eggs, d/kg

Q_F = quantity of feed (hay, mixed grains, corn silage) eaten by livestock, kg/d

C_F = concentration of contaminant in feed, μg/kg

f_F - fraction of contaminant in the feed that is absorbed by the animal, dimensionless

Q_W = quantity of water consumed by livestock, l/d

C_W = concentration of contaminant in water, mg/m^3

f_W = fraction of contaminant in water that is absorbed by the animal, dimensionless

Q_A = quantity of air breathed by livestock, m^3/d

C_A = concentration of contaminant in air, mg/m^3

f_a = fraction of contaminant in air that is absorbed by the animal, dimensionless

Q_S = quantity of soil consumed by livestock, kg/d

C_S = concentration of contaminant in soil, μg/kg

f_S = fraction of contaminant in soil that is absorbed by the animal, dimensionless

1000 = unit conversion factor

Dietary assumptions (Q_F, Q_W, Q_A and Q_S) used in the model are shown in Table D-36. Absorption factors (f_F,

TABLE D-36

TOTAL DIETARY ASSUMPTIONS FOR LIVESTOCK[1]

	Dairy Cow	Steer	Swine	Poultry
Hay[2] (kg/d)	5.98	1.14	NA	NA
Corn[2] (kg/d)	5.99	1.12	1.25	NA
Mixed grains[2] (kg/d)	5.02	6.78	1.25	0.07
Air (m^3/d)	125	125	8	1
Water (L/d)	60[3]	40[3]	8[3]	0.2[3]
Soil (kg/d)	1.08	0.72	NA	NA

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[1] All data from Ecologistics 1984a, 1986, unless otherwise noted.

[2] Dry weight

[3] National Research Council (NRC) 1974

f_W, f_A, f_S) were conservatively assumed to be 1.0 for all chemicals except PCDD/PCDF, which is assumed to be adsorbed to flyash due to the low temperatures in the post-combustion zone of the incinerator (see Appendix C). The values used for adsorption factors of PCDD/PCDF and for biotransfer factors are discussed in the following subsections.

D.6.2 BIOTRANSFER FACTORS

The biotransfer factors for metals retained in meat, milk, and eggs were based on studies conducted by Baes et al. (1984). For most organic chemicals, biotransfer factors for beef (F_b), milk (F_m), pork (F_p), poultry (F_c), and eggs (F_e) were estimated using the following correlations developed by Travis et al. (1987) and Baes (unpublished):

$$\log F_b = -7.60 + 1.00 (\log K_{ow})$$

$$\log F_m = -8.09 + 1.00 (\log K_{ow})$$

$$\log F_p = -4.80 + 0.62 (\log K_{ow})$$

$$\log F_e = -2.53 + 0.54 (\log K_{ow})$$

$$\log F_c = -2.59 + 0.54 (\log K_{ow})$$

Where:

F_b = fraction of the chemical consumed each day which is retained in beef, d/kg

F_m = fraction of the chemical consumed each day which is retained in milk, d/kg

F_p = fraction of the chemical consumed each day which is retained in pork, d/kg

F_e = fraction of the chemical consumed each day which is retained in eggs, d/kg

F_c = fraction of the chemical consumed each day which is retained in poultry, d/kg

K_{ow} = octanol/water partition coefficient

These correlations are based on the octanol-water partition coefficient (K_{ow}), which describes how a chemical will distribute itself between a water phase and a hydrophobic octanol phase. In this way, octanol partitioning is used as an estimate of the lipid solubility of a chemical, and hence how it will be accumulated in body fat. K_{ow} values for organic contaminants are presented in Table D-41 at the end of this appendix.

Experiments have been conducted to gain a better understanding of the potential for accumulation of PCDD/PCDF, PCBs, and hexachlorobenzene in beef and milk. For these chemicals, direct measurements of F_m and F_b have been used in the risk assessment. The derivation of F_m and F_b values for PCDD/PCDF is described in detail in Appendix F. The bases for the values used for PCBs and hexachlorobenzene are provided below.

PCBs - Milk. In a study conducted by Fries et al. (1973), nine dairy cows were fed 200 mg/d of Aroclor 1254. An examination of the average PCB concentrations in the milk fat of these cows indicates that steady-state conditions were apparently reached by the conclusion of the study (60 days). The average PCB concentration in the milk fat on day 60 was 60.9 ± 4.5 µg/g. Therefore the milk fat biotransfer factor can be estimated as:

$$B_{mf} = \frac{60.9 \text{ mg/kg}}{200 \text{ mg/d}} = 0.3 \text{ d/kg}$$

Assuming 4% fat in milk, the biotransfer factor for PCBs to milk is:

$$B_m = 0.3 \times 0.04 = 0.012 \text{ d/kg}$$

PCBs - Beef. In the Fries et al. (1973) study described above, PCB levels in tissue fat as well as milk fat were measured. At the end of the study (60 days), an average PCB concentration of 41.7 ± 11.5 µg/g was detected in tissue fat. It is not clear, however, that steady-state concentrations had been reached. Therefore, a single compartment elimination model approach was used to estimate steady state concentrations. (This approach is described in detail in Appendix F.) If initial contaminant concentrations are negligible, the single compartment model allows for the calculation of the steady-state concentration using the following equation:

$$C_{ss} = C/[(1 - \exp(-kt)]$$

Where:

C_{ss} = contaminant concentration at steady-state, mg/kg

C = contaminant concentration at the time of measurement, mg/kg

k = elimination rate constant, d^{-1}

t = period over which accumulation occurs, d

Based on the body fat dissipation study conducted by Fries et al., an elimination half-life of approximately 100 days was calculated. (This value is similar to the half-life estimated in Appendix F for PCDD/PCDF.) This half-life corresponds to an elimination rate constant (k) of 0.007 days^{-1}. Using the measured PCB concentration at day 60, 41.7 µg/g, a steady-state concentration of 122 µg/g was estimated. Therefore, the biotransfer factor for beef fat is:

$$B_{mf} = \frac{122 \text{ mg/kg}}{200 \text{ mg/d}} = 0.3 \text{ d/kg}$$

Assuming 25% fat in beef, the biotransfer factor for beef is:

$$B_b = 0.61 \times 0.25 = 0.15 \text{ d/kg}$$

Hexachlorobenzene - Milk. In a study conducted by Borzelleca et al. (1971), the bioconcentration of various chemicals, including hexachlorobenzene (HCB) in several animals, including lactating cows, was examined. In the study, daily HCB doses of 180 ppb, 18 ppb, and 1.8 ppb were administered. Each dose level was given to three cows, and the milk was analyzed for HCB periodically over a period of 56 days.

Although it is not clear from the data that steady-state HCB concentrations were reached during the 56 day study, levels do appear to be nearing a plateau. Furthermore, the 56 day exposure is very close to the 60 days period found by Fries et al. (1973) to be required for PCBs to reach steady-state concentrations in milk. Therefore, the highest concentrations measured in the tests involving 180 ppb and 18 ppb dose levels were used to calculate biotransfer factors. These concentrations are 15 ppb and 3 ppb, respectively. (HCB concentrations in milk from cows administered a dose of 1.8 ppb HCB were too low for estimating a biotransfer factor.)

To calculate biotransfer factors, a feed rate of 16 kg/d was assumed. Using this value, the following doses can be calculated:

$$180 \frac{\mu g}{kg} \times \frac{16\ kg}{d} = 2.88\ mg/d$$

$$18 \frac{\mu g}{kg} \times \frac{16\ kg}{d} = 0.288\ mg/d$$

The biotransfer factors corresponding to the two dose levels can then be calculated:

$$B_m = \frac{0.015\ mg/kg}{2.88\ mg/d} = 0.005\ d/kg$$

$$B_m = \frac{0.003\ mg/kg}{2.888\ mg/d} = 0.010\ d/kg$$

The average biotransfer factor is thus approximately 0.0075 d/kg.

A second study, conducted by Fries and Marrow (1976), also examined the accumulation of HCB in milk. In this study, a maximum concentration of approximately 0.4 mg/kg HCB was detected in milk fat for a dose of 1 mg/d. Fries and Marrow concluded that the HCB concentrations measured at 60 days were not necessarily steady-state levels, although "they do provide a reliable basis for establishing the dietary amounts that must be maintained to keep milk concentrations below regulatory guidelines".

In keeping with the conservative nature of this risk assessment, however, the single compartment model was used to predict steady-state HCB concentrations in milk. Based on an elimination rate constant of 0.0145, and 4 percent fat in milk, a B_m value of 0.028 d/kg was calculated. Since this value is higher than the one estimated based on the Borzelleca et al. (1971) data, it was used in the risk assessment.

Hexachlorobenzene - Beef. In the Borzelleca et al. (1971) study described above, contaminant concentrations in fat (both abdominal and subcutaneous), skeletal muscle, and other body tissues were measured. The data for HCB in the fat indicate that steady-state concentrations were not reached during the 112 day period over which tissue samples were collected. Furthermore, dissipation studies were not conducted, so that an elimination rate constant cannot be calculated on the basis of the Borzelleca et al. study.

In order to estimate steady state concentrations, the one compartment model approach described in Appendix F was used. For the purposes of this model, a dissipation rate constant (k) of 0.007 days^{-1} was assumed. This value, which corresponds to an elimination half-life of 100 days, is approximately that previously calculated for both PCDD/PCDF and PCBs.

The following results were used to estimate the extent of HCB biotransfer to body fat:

Dose Level (ppb)	Exposure Period (days)	HCB Concentration (ppm) Abdominal Fat	HCB Concentration (ppm) Subcutaneous Fat	HCB Concentration (ppm) Average
180	112	0.785	0.722	0.75
180	84	0.698	0.537	0.62
18	112	0.102	0.079	0.091
18	84	0.046	0.030	0.038
1.8	84	0.013	0.010	0.012

The steady-state concentration can be calculated using the one compartment model:

Dose Level (ppb)	$k(d^{-1})$	t(d)	C(ppm)	C_{ss}(ppm)
180	0.007	112	0.75	1.4
180	0.007	84	0.62	1.4
18	0.007	112	0.091	0.17
18	0.007	84	0.038	0.085
1.8	0.007	84	0.012	0.027

Biotransfer factors for beef fat can be calculated by assuming a 16 kg/d feed rate:

Dose Level (µg/kg)	Dose Level (mg/d)	C_{ss} (mg/kg)	Biotransfer factor, beef fat (d/kg)
180	2.88	1.4	0.49
180	2.88	1.4	0.49
18	0.288	0.17	0.59
18	0.288	0.085	0.30
1.8	0.0288	0.027	0.94

The average biotransfer factor to beef fat is thus 0.56 d/kg. The corresponding biotransfer factor to beef (assuming 25% fat in beef) is $B_b = 0.14$ d/kg.

A second study examining the accumulation of HCB in beef fat has also been conducted (Fries and Marrow 1976). In this study, a concentration of approximately 0.37 mg/kg HCB was dectected in body fat per 1 mg/d HCB administered. Based on an assumed dissiptation rate constant of 0.007 days^{-1}, a steady-state B_m value of 0.27 d/kg was calculated. Since this value is higher than the one estimated based on the Borzelleca et al. (1971) data, it was used in the risk assessment.

It should be recognized that considerable uncertainty may exist in those biotransfer factors based on the generalized correlations rather than actual measurements. This is particularly true for estimates of the biotransfer of contaminants to pork, poultry, and eggs, where data are especially sparse. The values of F_b, F_m, F_p, F_e, and F_c for the chemicals evaluated in the risk assessment are shown in Table D-37.

TABLE D-37

BIOTRANSFER FACTORS

CHEMICAL CONSTITUENT	FRACTION RETAINED IN BEEF (Fb)	FRACTION RETAINED IN MILK (Fm)	FRACTION RETAINED IN PORK (Fp)	FRACTION RETAINED IN POULTRY (Fc)	FRACTION RETAINED IN EGGS (Fe)
ACETONE	1.45E-08	4.68E-09	1.13E-05	1.91E-03	2.19E-03
ACRYLONITRILE	4.47E-08	1.45E-08	2.27E-05	3.51E-03	4.03E-03
BENZENE	3.39E-06	1.10E-06	3.32E-04	3.63E-02	4.17E-02
BENZO(A)ANTHRACENE	1.03E-02	3.33E-03	4.79E-02	2.76E+00	3.17E+00
BENZO(A)PYRENE	2.89E-02	9.35E-03	9.07E-02	4.82E+00	5.53E+00
BROMODICHLOROMETHANE	3.16E-06	1.02E-06	3.18E-04	3.50E-02	4.02E-02
BROMOFORM	6.03E-06	1.95E-06	4.74E-04	4.96E-02	5.69E-02
BUTADIENE,1,3-	2.45E-06	7.94E-07	2.72E-04	3.05E-02	3.50E-02
CARBON TETRACHLORIDE	1.70E-05	5.49E-06	9.01E-04	8.67E-02	9.96E-02
CHLOROBENZENE	1.73E-05	5.61E-06	9.12E-04	8.77E-02	1.01E-01
CHLOROFORM	2.34E-06	7.59E-07	2.64E-04	2.98E-02	3.42E-02
DICHLORODIFLUOROMETHANE	3.01E-06	9.75E-07	3.08E-04	3.41E-02	3.92E-02
DIOXIN,2,3,7,8-EQ(EPA)	3.30E-01	2.20E-02	2.04E-01	9.78E+00	1.12E+01
DIOXIN,2,3,7,8-EQ(MOE)	5.40E-02	4.40E-03	2.04E-01	9.78E+00	1.12E+01
ETHANOL	1.20E-08	3.89E-09	1.00E-05	1.73E-03	1.98E-03
ETHYLENE GLYCOL	2.95E-10	9.55E-11	1.01E-06	2.33E-04	2.68E-04
ETHYLENE OXIDE	1.26E-08	4.06E-09	1.03E-05	1.77E-03	2.03E-03
FORMALDEHYDE	5.62E-09	1.82E-09	6.27E-06	1.15E-03	1.32E-03
HEXACHLOROBENZENE	2.70E-01	2.80E-02	1.50E-01	7.48E+00	8.59E+00
HYDRAZINE	1.07E-09	3.47E-10	2.24E-06	4.68E-04	5.37E-04
METHYL ETHYL KETONE	4.57E-08	1.48E-08	2.30E-05	3.55E-03	4.08E-03
METHYLENE CHLORIDE	4.57E-07	1.48E-07	9.58E-05	1.23E-02	1.41E-02
NAPHTHALENE	4.90E-05	1.59E-05	1.74E-03	1.54E-01	1.76E-01
NITROPHENOL,o-	1.41E-06	4.57E-07	1.93E-04	2.26E-02	2.60E-02
PCB	1.50E-01	1.12E-02	8.81E-02	4.69E+00	5.39E+00
PETROLEUM DISTILLATES	1.17E-07	3.80E-08	4.12E-05	5.91E-03	6.79E-03
PHENOL	7.24E-07	2.34E-07	1.27E-04	1.58E-02	1.81E-02
PYRIDINE	1.10E-07	3.55E-08	3.95E-05	5.70E-03	6.54E-03
STYRENE	2.24E-05	7.24E-06	1.07E-03	1.01E-01	1.16E-01
TETRACHLOROETHYLENE	1.00E-05	3.24E-06	6.49E-04	6.52E-02	7.48E-02
TETRAETHYL LEAD	9.33E-04	3.02E-04	1.08E-02	7.55E-01	8.67E-01
TOLUENE	1.23E-05	3.98E-06	7.38E-04	7.29E-02	8.37E-02
TRICHLOROETHANE,1,1,1-	8.13E-06	2.63E-06	5.70E-04	5.83E-02	6.69E-02
TRICHLOROETHYLENE	5.61E-06	2.14E-06	5.02E-04	5.21E-02	5.98E-02
TRICHLOROTRIFLUOROETHANE	6.17E-05	2.00E-05	2.00E-03	1.74E-01	2.00E-01
XYLENE	3.02E-05	9.77E-06	1.29E-03	1.18E-01	1.36E-01
ARSENIC	2.00E-03	6.00E-05	2.00E-03	2.00E-03	2.00E-03
BERYLLIUM	1.00E-03	9.00E-07	1.00E-03	1.00E-03	1.00E-03
CADMIUM	5.50E-04	1.00E-03	5.50E-04	5.50E-04	5.50E-04
CHROMIUM (VI)	5.50E-03	1.50E-03	5.50E-03	5.50E-03	5.50E-03
COPPER	1.00E-02	1.50E-03	1.00E-02	1.00E-02	1.00E-02
LEAD	3.00E-04	2.50E-04	3.00E-04	3.00E-04	3.00E-04
MERCURY	2.50E-01	4.50E-04	2.50E-01	2.50E-01	2.50E-01
NICKEL	6.00E-03	1.00E-03	6.00E-03	6.00E-03	6.00E-03
ZINC	1.00E-01	1.00E-02	1.00E-01	1.00E-01	1.00E-01

D.6.3 BIOAVAILABILITY OF PCDD/PCDF

In evaluating the adsorption behavior of PCDD/PCDF, it has been assumed that the majority of PCDD/PCDF emitted from the incinerator will be bound to flyash. Emphasis has therefore been placed on bioavailability of PCDD/PCDF associated with particles, in particular incinerator flyash. The bioavailability of PCDD/PCDF adsorbed onto incinerator flyash has been examined in several laboratory experiments (see, for example Van den Berg et al. 1983; Van den Berg et al. 1986; Van den Berg et al. 1987). In general, the bioavailability of PCDD/PCDF adsorbed onto flyash particles has been shown to be approximately ten times lower than that for PCDD/PCDF administered in an oil vehicle (Van den Berg et al. 1987). It has been hypothesized that the reduced bioavailability is a result of the strong chemical and/or physical binding of PCDD/PCDF to the surface of particles formed at high temperatures (Shaub 1985).

Data from two studies have been reviewed in Phase 4B to develop an estimate of the bioavailability of PCDD/PCDF bound to particles (Van den Berg 1983, Van den Berg 1987). In these experiments, laboratory rats were administered PCDD/PCDF both adsorbed to incinerator flyash and as an extract. The resulting concentrations of PCDD/PCDF in the liver of each rat were measured, thus enabling an evaluation of the relative bioavailability of particulate bound PCDD/PCDF.

The following conclusions can be drawn based on the two experiments conducted by Van den Berg:

- PCDD/PCDF substituted with chlorine in the 2, 3, 7, and 8 positions are selectively retained in the liver of an exposed rat;
- The relative bioavailability of tetrachlorinated CDD and CDF (TCDD/TCDF) bound to flyash is higher than that for PCDD/PCDF containing five or six chlorine atoms (PnCDD/PnCDF, HxCDD/HxCDF).

In the experiments, the observed absorption of particulate-bound TCDD/TCDF ranged from less than 15 percent to approximately 32 percent of that for unbound TCDD/TCDF. The bioavailability of particulate bound PnCDD/PnCDF and HxCDD/HxCDF relative to the unbound extract ranged from less than 3 percent to approximately 10 percent. In general, the relative bioavailability of TCDD/TCDF appears to be about 25 percent; for PnCDD/PnCDF and HxCDD/HxCDF, a relative absorption of approximately 10 to 15 percent was indicated.

Although it has been assumed that the majority of PCDD/PCDF emitted from the incinerator will be bound to flyash, it is possible that some fraction will be released in the vapor phase. This vapor phase PCDD/PCDF would be expected to sorb to available surfaces in the environment, e.g., soil, plants, and airborne particulate matter. A study of the gastrointestinal absorption of PCDD/PCDF sorbed to soil has been performed by Poiger and Schlatter (1980). The results of this investigation indicate that the relative absorption of such PCDD/PCDF may be on the order of 50 percent.

Based on the assumption that the PCDD/PCDF emitted from the incinerator will be adsorbed to flyash, an upperbound value for relative absorption in the gastrointestinal tract is approximately 25 percent. Therefore, f_F and f_S for PCDD/PCDF were assumed to be 0.25, since the PCDD/PCDF on plants and in soil is expected to be bound to flyash. (Root uptake of PCDD/PCDF by plants is low relative to the contribution from direct deposition). The values for f_W and f_a were conservatively assumed to be 1.0.

D.6.4 ESTIMATED CHEMICAL CONCENTRATIONS

Chemical concentrations in meat, milk, and eggs calculated using the approach described in this section are presented in Tables D-38, D-39, and D-40. These tables correspond to incinerator, evaporator, and fugitive emissions, respectively.

TABLE D-38

CONCENTRATIONS OF CHEMICALS IN MEAT, MILK AND EGGS - INCINERATOR EMISSIONS

CHEMICAL CONSTITUENT	BEEF CONC (ug/kg)	MILK CONC (ug/kg)	PORK CONC (ug/kg)	POULTRY CONC (ug/kg)	EGG CONC (ug/kg)
ACETONE	3.94E-07	5.36E-07	1.41E-04	1.24E-04	1.42E-04
ACRYLONITRILE	3.59E-09	4.83E-09	8.27E-07	6.70E-07	7.69E-07
BENZENE	5.28E-07	5.73E-07	1.74E-05	1.56E-05	1.80E-05
BENZO(A)ANTHRACENE	1.59E-03	7.43E-04	1.33E-03	3.13E-03	3.60E-03
BENZO(A)PYRENE	1.86E-03	8.80E-04	1.03E-03	2.20E-03	2.53E-03
BROMODICHLOROMETHANE	3.31E-08	3.80E-08	1.20E-06	8.73E-07	1.00E-06
BROMOFORM	1.53E-08	1.74E-08	4.18E-07	2.52E-07	2.90E-07
BUTADIENE,1,3-	3.07E-10	2.55E-10	8.40E-09	1.77E-08	2.04E-08
CARBON TETRACHLORIDE	2.87E-07	2.61E-07	3.87E-06	4.07E-06	4.67E-06
CHLOROBENZENE	9.56E-08	9.67E-08	1.44E-06	8.89E-07	1.02E-06
CHLOROFORM	7.19E-07	8.14E-07	2.90E-05	2.43E-05	2.79E-05
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	2.54E-06	2.36E-07	2.63E-07	5.19E-07	5.96E-07
DIOXIN,2,3,7,8-EQ(MOE)	8.63E-06	9.80E-07	5.46E-06	1.08E-05	1.24E-05
ETHANOL	5.42E-07	1.85E-07	2.09E-04	1.86E-04	2.14E-04
ETHYLENE GLYCOL	2.50E-07	5.20E-08	2.70E-04	1.22E-03	1.40E-03
ETHYLENE OXIDE	1.79E-10	6.10E-11	6.78E-08	5.99E-08	6.88E-08
FORMALDEHYDE	2.30E-09	7.88E-10	1.19E-06	1.12E-06	1.29E-06
HEXACHLOROBENZENE	4.24E-02	3.88E-03	8.89E-03	3.58E-03	4.11E-03
HYDRAZINE	1.67E-13	5.71E-14	1.62E-10	1.74E-10	1.99E-10
METHYL ETHYL KETONE	1.91E-07	6.48E-08	4.38E-05	3.53E-05	4.05E-05
METHYLENE CHLORIDE	2.40E-07	7.88E-08	2.00E-05	1.81E-05	2.08E-05
NAPHTHALENE	9.40E-06	2.22E-06	7.65E-05	4.15E-05	4.76E-05
NITROPHENOL,o-	1.78E-07	5.59E-08	9.75E-06	6.45E-06	7.40E-06
PCB	7.07E-03	2.52E-04	5.69E-04	8.54E-04	9.81E-04
PETROLEUM DISTILLATES	1.94E-07	6.50E-08	2.82E-05	2.79E-05	3.21E-05
PHENOL	2.90E-05	1.41E-05	1.02E-03	3.16E-03	3.63E-03
PYRIDINE	4.58E-08	1.53E-08	7.38E-06	5.72E-06	6.56E-06
STYRENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	6.67E-06	1.90E-06	1.28E-04	1.07E-04	1.23E-04
TETRAETHYL LEAD	9.35E-07	1.56E-07	9.28E-07	8.00E-07	9.19E-07
TOLUENE	2.42E-05	6.74E-06	4.33E-04	3.04E-04	3.49E-04
TRICHLOROETHANE,1,1,1-	2.14E-06	6.26E-07	4.24E-05	4.72E-05	5.42E-05
TRICHLOROETHYLENE	3.00E-06	8.83E-07	6.95E-05	6.53E-05	7.50E-05
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
XYLENE	2.91E-05	7.42E-06	3.07E-04	2.16E-04	2.48E-04
ARSENIC	2.42E-01	1.09E-02	4.83E-02	1.21E-03	1.21E-03
BERYLLIUM	1.09E-05	1.34E-08	1.74E-06	7.63E-08	7.63E-08
CADMIUM	3.47E-02	1.04E-01	7.43E-03	1.97E-04	1.97E-04
CHROMIUM (VI)	2.44E+00	9.90E-01	4.79E-01	1.27E-02	1.27E-02
COPPER	5.19E+00	1.18E+00	1.06E+00	2.93E-02	2.93E-02
LEAD	2.80E-01	3.43E-01	5.06E-02	1.72E-03	1.72E-03
MERCURY	6.77E+00	1.93E-02	1.50E+00	5.39E-02	5.39E-02
NICKEL	1.76E+00	4.38E-01	3.50E-01	8.98E-03	8.98E-03
ZINC	3.90E+02	7.24E+01	9.72E+01	2.68E+00	2.68E+00

TABLE D-39

CONCENTRATIONS OF CHEMICALS IN MEAT, MILK AND EGGS - EVAPORATOR EMISSIONS

CHEMICAL CONSTITUENT	BEEF CONC (ug/kg)	MILK CONC (ug/kg)	PORK CONC (ug/kg)	POULTRY CONC (ug/kg)	EGG CONC (ug/kg)
ACETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ACRYLONITRILE	4.78E-08	6.44E-08	1.10E-05	8.92E-06	1.02E-05
BENZENE	4.03E-08	4.38E-08	1.33E-06	1.19E-06	1.36E-06
BENZO(A)ANTHRACENE	5.17E-05	2.42E-05	4.33E-05	1.02E-04	1.17E-04
BENZO(A)PYRENE	4.24E-04	2.01E-04	2.36E-04	5.04E-04	5.79E-04
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.23E-09	1.85E-09	6.10E-08	1.29E-07	1.48E-07
CARBON TETRACHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	3.46E-07	1.36E-07	3.16E-06	2.95E-05	3.38E-05
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
FORMALDEHYDE	5.28E-11	7.20E-11	2.73E-08	2.57E-08	2.95E-08
HEXACHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NAPHTHALENE	4.56E-04	4.17E-04	3.72E-03	1.92E-03	2.21E-03
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	1.64E-04	2.14E-05	1.30E-05	1.96E-05	2.25E-05
PETROLEUM DISTILLATES	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PHENOL	2.91E-05	1.42E-05	1.03E-03	3.16E-03	3.63E-03
PYRIDINE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
STYRENE	5.95E-07	5.82E-07	7.65E-06	4.92E-06	5.64E-06
TETRACHLOROETHYLENE	4.55E-07	4.62E-07	8.77E-06	7.21E-06	8.28E-06
TETRAETHYL LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TOLUENE	4.85E-06	5.01E-06	8.70E-05	5.99E-05	6.87E-05
TRICHLOROETHANE,1,1,1-	4.21E-06	4.02E-06	8.31E-05	9.28E-05	1.07E-04
TRICHLOROETHYLENE	1.17E-07	1.19E-07	2.71E-06	2.52E-06	2.90E-06
TRICHLOROTRIFLUOROETHAN	8.42E-06	3.79E-06	2.69E-05	1.54E-04	1.77E-04
XYLENE	2.18E-06	2.03E-06	2.31E-05	1.58E-05	1.82E-05
ARSENIC	2.40E-01	1.08E-02	4.80E-02	1.20E-03	1.20E-03
BERYLLIUM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CADMIUM	2.20E-02	6.00E-02	4.40E-03	1.10E-04	1.10E-04
CHROMIUM (VI)	2.20E+00	9.00E-01	4.40E-01	1.10E-02	1.10E-02
COPPER	4.00E+00	9.00E-01	8.00E-01	2.00E-02	2.00E-02
LEAD	1.08E-01	1.35E-01	2.16E-02	5.40E-04	5.40E-04
MERCURY	4.00E-01	1.08E-03	8.00E-02	2.00E-03	2.00E-03
NICKEL	1.68E+00	4.20E-01	3.36E-01	8.40E-03	8.40E-03
ZINC	6.80E+01	1.02E+01	1.36E+01	3.40E-01	3.40E-01

TABLE D-40

CONCENTRATIONS OF CHEMICALS IN MEAT, MILK AND EGGS - FUGITIVE EMISSIONS

CHEMICAL CONSTITUENT	BEEF CONC (ug/kg)	MILK CONC (ug/kg)	PORK CONC (ug/kg)	POULTRY CONC (ug/kg)	EGG CONC (ug/kg)
ACETONE	1.11E-05	2.87E-05	7.59E-03	6.64E-03	7.62E-03
ACRYLONITRILE	3.20E-07	4.31E-07	7.38E-05	5.97E-05	6.86E-05
BENZENE	5.48E-06	5.97E-06	1.81E-04	1.62E-04	1.85E-04
BENZO(A)ANTHRACENE	8.24E-12	3.85E-12	6.91E-12	1.63E-11	1.87E-11
BENZO(A)PYRENE	9.59E-12	4.55E-12	5.34E-12	1.14E-11	1.31E-11
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	4.59E-07	3.89E-07	1.28E-05	2.71E-05	3.11E-05
CARBON TETRACHLORIDE	9.04E-06	8.27E-06	1.22E-04	1.27E-04	1.46E-04
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	5.46E-06	6.20E-06	2.20E-04	1.83E-04	2.11E-04
DICHLORODIFLUOROMETHANE	8.92E-07	3.51E-07	8.14E-06	7.59E-05	8.72E-05
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	1.37E-05	1.86E-05	5.26E-03	4.70E-03	5.39E-03
ETHYLENE GLYCOL	3.12E-07	2.59E-07	3.37E-04	1.52E-03	1.75E-03
ETHYLENE OXIDE	3.70E-09	5.04E-09	1.40E-06	1.24E-06	1.42E-06
FORMALDEHYDE	3.96E-06	5.27E-06	1.99E-03	1.88E-03	2.16E-03
HEXACHLOROBENZENE	5.61E-07	2.05E-07	1.18E-07	4.73E-08	5.43E-08
HYDRAZINE	3.24E-12	4.44E-12	3.16E-09	3.38E-09	3.88E-09
METHYL ETHYL KETONE	5.98E-06	8.06E-06	1.37E-03	1.10E-03	1.26E-03
METHYLENE CHLORIDE	2.31E-05	2.30E-05	1.93E-03	1.74E-03	1.99E-03
NAPHTHALENE	3.22E-08	2.94E-08	2.62E-07	1.35E-07	1.56E-07
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	6.85E-04	8.95E-05	5.42E-05	8.19E-05	9.43E-05
PETROLEUM DISTILLATES	3.07E-06	3.32E-06	4.46E-04	4.41E-04	5.03E-04
PHENOL	2.96E-05	1.48E-05	1.36E-03	3.19E-03	3.67E-03
PYRIDINE	8.25E-07	1.10E-06	1.33E-04	1.03E-04	1.18E-04
STYRENE	8.07E-07	7.90E-07	1.04E-05	6.66E-06	7.63E-06
TETRACHLOROETHYLENE	1.17E-04	1.19E-04	2.26E-03	1.86E-03	2.13E-03
TETRAETHYL LEAD	1.38E-05	8.47E-06	1.31E-05	1.05E-05	1.20E-05
TOLUENE	6.37E-04	6.57E-04	1.14E-02	7.87E-03	9.03E-03
TRICHLOROETHANE,1,1,1-	6.34E-05	6.10E-05	1.26E-03	1.39E-03	1.60E-03
TRICHLOROETHYLENE	8.01E-05	8.19E-05	1.86E-03	1.73E-03	1.99E-03
TRICHLOROTRIFLUOROETHANE	3.37E-06	1.52E-06	1.07E-05	6.17E-05	7.09E-05
XYLENE	4.61E-04	4.31E-04	4.39E-03	3.35E-03	3.84E-03

TABLE D-41

PHYSICAL/CHEMICAL PROPERTIES FOR ORGANIC CONTAMINANTS

CHEMICAL CONSTITUENT	VAPOR PRESSURE AT 25C (mm Hg)	REFERENCE	LOG Kow	REFERENCE
ACETONE	2.34E+02	Verscheuren 1983	-0.24	Verscheuren 1983
ACRYLONITRILE	1.00E+02	Verscheuren 1983	0.25	Mabey 1982
BENZENE	9.45E+01	Reid 1977	2.13	Mabey 1982
BENZO(A)ANTHRACENE	5.00E-09	Mabey 1982	5.61	Mabey 1982
BENZO(A)PYRENE	5.60E-09	Mabey 1982	6.06	Mabey 1982
BROMODICHLOROMETHANE	5.00E+01	Mabey 1982	2.10	Mabey 1982
BROMOFORM	5.00E+00	Mabey 1982	2.38	Mabey 1982
BUTADIENE,1,3-	2.10E+03	Reid 1977	1.99	Hansch and Leo 1979
CARBON TETRACHLORIDE	1.13E+02	Verscheuren 1983	2.83	Hansch and Leo 1979
CHLOROBENZENE	1.18E+01	Verscheuren 1983	2.84	Mabey 1982
CHLOROFORM	1.93E+02	Reid 1977	1.97	Verscheuren 1983
DICHLORODIFLUOROMETHANE	4.25E+03	Verscheuren 1983	2.08	Mabey 1982
DIOXIN,2,3,7,8-EQ(EPA)	7.40E-10	Podoll 1986	6.63	Marple 1986a
DIOXIN,2,3,7,8-EQ(MOE)	7.40E-10	Podoll 1986	6.63	Marple 1986a
ETHANOL	5.86E+01	Reid 1977	-0.32	Versheuren 1983
ETHYLENE GLYCOL	1.25E-01	Verscheuren 1983	-1.93	Verscheuren 1983
ETHYLENE OXIDE	1.10E+03	Verscheuren 1983	-0.30	Versar 1984
FORMALDEHYDE	3.83E+03	Reid 1977	-0.65	Hansch and Leo 1979
HEXACHLOROBENZENE	1.68E-05	Calculated	6.41	Mabey 1982
HYDRAZINE	1.60E+01	Verscheuren 1983	-1.370	Verscheuren 1983
METHYL ETHYL KETONE	1.00E+02	Reid 1977	0.26	Verscheuren 1983
METHYLENE CHLORIDE	4.33E+02	Reid 1977	1.26	Mabey 1982
NAPHTHALENE	9.40E-02	Calculated	3.29	Mabey 1982
NITROPHENOL,o-	1.51E-01	Mabey 1982	1.75	Mabey 1982
PCB	7.71E-05	Mabey 1982	6.04	Mabey 1982
PETROLEUM DISTILLATES	4.00E+00	GE Manual 1980	0.67	Calculated
PHENOL	5.00E-01	Verscheuren 1983	1.46	Versheuren 1983
PYRIDINE	2.06E+01	Reid 1977	0.64	Calculated
STYRENE	6.12E+00	Reid 1977	2.95	Lyman 1982
TETRACHLOROETHYLENE	1.40E+01	Mabey 1982	2.60	Verscheuren 1983
TETRAETHYL LEAD	1.52E-01	Verscheuren 1983	4.57	Bradlaw 1980
TOLUENE	2.82E+01	Reid 1977	2.69	Versheuren 1983
TRICHLOROETHANE,1,1,1-	1.00E+02	Verscheuren 1983	2.51	Mabey 1982
TRICHLOROETHYLENE	7.38E+01	Reid 1977	2.42	Mabey 1982
TRICHLOROTRIFLUOROETHANE	3.32E+02	Reid 1977	3.39	Calculated
XYLENE	5.80E+00	Versheuren	3.08	Verscheuren 1983

continued ...

TABLE D-41 (continued)

PHYSICAL/CHEMICAL PROPERTIES FOR ORGANIC CONTAMINANTS

CHEMICAL CONSTITUENT	Koc	REFERENCE	SOLUBILITY AT 25C (mg/l)	REFERENCE
ACETONE	1.76E+01	Calculated	6.01E+05	Calculated
ACRYLONITRILE	8.50E-01	Mabey 1982	7.90E+04	Mabey 1982
BENZENE	6.50E+01	Mabey 1982	1.78E+03	Mabey 1982
BENZO(A)ANTHRACENE	2.00E+05	Mabey 1982	5.70E-03	Mabey 1982
BENZO(A)PYRENE	5.50E+06	Mabey 1982	3.80E-03	Mabey 1982
BROMODICHLOROMETHANE	6.10E+01	Mabey 1982	4.50E+03	Mabey 1982
BROMOFORM	1.16E+02	Mabey 1982	3.03E+03	Mackay & Shui 1981
BUTADIENE,1,3-	1.16E+02	Calculated	7.35E+02	Verscheuren 1983
CARBON TETRACHLORIDE	4.39E+02	Mabey 1982	1.16E+03	Verscheuren 1983
CHLOROBENZENE	3.30E+02	Mabey 1982	4.88E+02	Mabey 1982
CHLOROFORM	4.40E+01	Mabey 1982	9.30E+03	Verscheuren 1983
DICHLORODIFLUOROMETHANE	5.80E+01	Mabey 1982	2.80E+02	Verscheuren 1983
DIOXIN,2,3,7,8-EQ(EPA)	1.71E+06	Calculated	1.93E-05	Marple 1986b
DIOXIN,2,3,7,8-EQ(MOE)	1.71E+06	Calculated	1.93E-05	Marple 1986b
ETHANOL	1.60E+01	Calculated	1.00E+06	Calculated
ETHYLENE GLYCOL	2.12E+02	Calculated	7.36E+07	Calculated
ETHYLENE OXIDE	1.64E+01	Versar 1984	2.10E+06	Versar 1984
FORMALDEHYDE	1.06E+01	Calculated	6.00E+05	Versar 1984
HEXACHLOROBENZENE	1.20E+06	Mabey 1982	6.00E-03	Mabey 1982
HYDRAZINE	4.30E+00	Calculated	1.00E+06	Calculated
METHYL ETHYL KETONE	1.00E-02	Calculated	3.53E+05	Verscheuren 1983
METHYLENE CHLORIDE	8.80E+00	Mabey 1982	1.67E+04	Verscheuren 1983
NAPHTHALENE	9.40E+02	Mabey 1982	3.00E+01	Perry & Chilton 1973
NITROPHENOL,o-	2.70E+01	Mabey 1982	2.10E+03	Verscheuren 1983
PCB	5.30E+05	Mabey 1982	3.10E-02	Mabey 1982
PETROLEUM DISTILLATES	1.60E+04	Calculated	9.34E-02	Verscheuren 1983
PHENOL	1.42E+01	Mabey 1982	9.30E+04	Callahan 1979
PYRIDINE	2.65E-02	Donaldson 1975	3.00E+08	Versar 1984
STYRENE	2.70E+02	Calculated	3.00E+02	Verscheuren 1983
TETRACHLOROETHYLENE	3.64E+02	Mabey 1982	1.50E+02	Verscheuren 1983
TETRAETHYL LEAD	7.30E+03	Calculated	3.10E+01	Verscheuren 1983
TOLUENE	3.00E+02	Mabey 1982	5.15E+02	Verscheuren 1983
TRICHLOROETHANE,1,1,1-	1.52E+02	Mabey 1982	4.40E+03	Verscheuren 1983
TRICHLOROETHYLENE	1.26E+02	Mabey 1982	1.10E+03	Callahan 1979
TRICHLOROTRIFLUOROETHANE	3.23E+02	Calculated	2.80E+02	GE Manual
XYLENE	2.46E+02	Calculated	1.87E+02	Verscheuren 1983

continued ...

TABLE D-41 (continued)

PHYSICAL/CHEMICAL PROPERTIES FOR ORGANIC CONTAMINANTS

CHEMICAL CONSTITUENT	DIFF IN AIR (cm^2/sec)	REFERENCE	HENRY'S LAW CONSTANT (unitless)	REFERENCE
ACETONE	1.05E-01	Lugg 1968	1.25E-03	Calculated
ACRYLONITRILE	1.06E-01	Lugg 1968	3.60E-03	Mabey 1982
BENZENE	9.32E-02	Lugg 1968	2.25E-01	Mabey 1982
BENZO(A)ANTHRACENE	5.30E-02	Calculated	4.17E-05	Mabey 1982
BENZO(A)PYRENE	5.16E-02	Calculated	2.04E-05	Mabey 1982
BROMODICHLOROMETHANE	8.60E-02	Calculated	1.00E-01	Mabey 1982
BROMOFORM	7.67E-02	Lugg 1968	2.33E-02	Mabey 1982
BUTADIENE,1,3-	3.19E-02	Calculated	8.47E+00	Calculated
CARBON TETRACHLORIDE	8.28E-02	Lugg 1968	1.24E+00	Gossett 1987
CHLOROBENZENE	7.47E-02	Lugg 1968	1.46E-01	Mabey 1982
CHLOROFORM	8.88E-02	Lugg 1968	1.50E-01	Gossett 1987
DICHLORODIFLUOROMETHANE	9.40E-02	Calculated	1.24E+02	Mabey 1986
DIOXIN,2,3,7,8-EQ(EPA)	5.00E-02	Calculated	6.67E 04	Calculated
DIOXIN,2,3,7,8-EQ(MOE)	5.00E-02	Calculated	6.67E 04	Calculated
ETHANOL	1.18E-01	Lugg 1968	1.46E 04	Calculated
ETHYLENE GLYCOL	1.01E-01	Lugg 1968	5.74E 09	Calculated
ETHYLENE OXIDE	1.31E-02	Calculated	1.48E-03	Calculated
FORMALDEHYDE	1.70E-01	Calculated	1.15E-02	Calculated
HEXACHLOROBENZENE	5.90E-02	Calculated	2.78E-02	Mabey 1982
HYDRAZINE	1.90E-01	Calculated	5.40E 07	Calculated
METHYL ETHYL KETONE	9.03E-02	Lugg 1968	1.50E-03	Mackay 1982
METHYLENE CHLORIDE	1.04E-01	Lugg 1968	8.95E-02	Gossett 1987
NAPHTHALENE	7.00E-02	Calculated	1.88E-02	Mabey 1982
NITROPHENOL,o-	3.31E-02	Calculated	3.15E-04	Mabey 1982
PCB	4.00E-02	Calculated	1.08E 01	Mabey 1982
PETROLEUM DISTILLATES	7.57E-02	Calculated	2.38E+02	Calculated
PHENOL	8.42E-02	Shen 1980	1.86E-05	Mabey 1982
PYRIDINE	9.50E-02	Calculated	2.90E-07	Calculated
STYRENE	7.04E-02	Shen 1980	1.87E-01	Calculated
TETRACHLOROETHYLENE	7.97E-02	Lugg 1968	7.23E-01	Gossett 1987
TETRAETHYL LEAD	1.00E-02	Calculated	3.28E+00	Lyman 1982
TOLUENE	8.49E-02	Lugg 1968	2.78E-01	Mabey 1982
TRICHLOROETHANE,1,1,1-	7.94E-02	Lugg 1968	7.03E-01	Gossett 1987
TRICHLOROETHYLENE	8.75E-02	Lugg 1968	3.92E-01	Gossett 1987
TRICHLOROTRIFLUOROETHANE	7.49E-02	Calculated	1.22E+01	Calculated
XYLENE	6.95E-02	Lugg 1968	2.20E-01	Lyman 1982

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Final report

**Ontario
Waste Management
Corporation**

A provincial crown corporation

ENVIRON

[illegible]

SITE ASSESSMENT
PHASE 4B: RISK ASSESSMENT
VOLUME 2
APPENDICES E THROUGH N

Prepared for
The Ontario Waste Management Corporation

Submitted by

ENVIRON Corporation

January, 1988

APPENDIX E

TRANSPORT OF CONTAMINANTS RELEASED INTO A STREAM

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APPENDIX E

TRANSPORT OF CONTAMINANTS RELEASED INTO A STREAM

E.1 INTRODUCTION

The local preferred access route for the proposed OWMC facility crosses several streams, including Twenty Mile Creek, Ten Mile Creek, and Five Mile Creek. A transportation accident which occurs in proximity to any of these crossings may result in a spill of potentially hazardous waste into a stream. In the Phase 4B risk assessment, the risks associated with a spill into Twenty Mile Creek were evaluated. Twenty Mile Creek was selected for study because it is the largest stream along the local access route and has a number of sensitive downstream uses. In Appendix B, the procedure used to select surrogate chemicals for the analysis of a spill into Twenty Mile Creek is described. In Appendix C, the spill scenario is characterized in terms of location, magnitude, and frequency. In this Appendix, approaches used to (a) describe the characteristics of Twenty Mile Creek and (b) predict the fate and transport of chemicals spilled into the stream are discussed.

E.2 CHARACTERIZATION OF TWENTY MILE CREEK

E.2.1 TOPOGRAPHY

From its source located near the city of Hamilton, Twenty Mile Creek meanders eastward in a shallow valley. The creek crosses beneath Regional Road 24, which is part of the preferred access route, at kilometer point 8.0, approximately 1.6 kilometers before reaching the escarpment at Ball's Falls, where it drops approximately 26 meters into a narrow gorge. At this point, the creek turns northwards and flows across a plain for a distance of about 4.8 kilometers to a large bay on Lake Ontario at Jordan Harbor. The distance from Regional Road 24, the point considered in the spill scenario, to the bay at Jordan Harbor is approximately 6.4 kilometers. The transport of chemicals spilled into Twenty Mile Creek at Regional Road 24 downstream toward the bay was modeled in the risk assessment. Topographical information for this section of Twenty Mile Creek is presented in Table E-1.

E.2.2 HYDROLOGY

The median flow of Twenty Mile Creek was chosen to represent the flow condition at the time of the spill. In order to estimate this flow, monthly and daily discharge records from the gauging station located at Ball's Falls were analyzed

TABLE E-1

TOPOGRAPHICAL INFORMATION FOR TWENTY MILE CREEK

Distance from the Bay (Km)	Elevation (m)	Slope
8.3	167	0.01107
6.2	144	0.02828
3.8	76	0.00232
0	75	

Source: Ontario Department of Energy and Resources Management, 1964.

[1] Regional Road 24 crosses Twenty Mile Creek about at kilometer point 8.0. Ball's Falls is a kilometer point 6.4. The concentrations in the creek were modeled from Regional Road 24 to the beginning of the bay, kilometer point 1.6.

(Water Survey of Canada 1985). Monthly average discharge records were available from 1957 to 1984. The monthly average flows for this period are summarized in Table E-2. As indicated in the table, Twenty Mile Creek's discharge drops quickly from a maximum in springtime to moderate flows in early summer and often to zero or nearly zero for extended periods in the late summer and early fall. In addition, according to the discharge records, icy conditions are often present in the winter.

Daily discharge records were available from 1976 to 1981. By comparing the monthly average flows of the years 1976 to 1981 to the monthly average flows of the years 1957 to 1984, it was determined that 1978 represented the year when the flow was nearest to the average flow or typical flow year. Therefore, the daily discharge records for the year 1978 were analyzed to determine the median flow for the year.

The frequency of daily discharge for the year 1978 is presented in Table E-3. The median flow, 0.42 cubic meters per second, was chosen to represent the condition of the stream at the time of the spill.

E.2.3 HYDRAULICS

A chemical spilled into Twenty Mile Creek will be advected downstream toward Jordan Harbor. Since velocity measurements

TABLE E-2

TWENTY MILE CREEK FLOW DATA (m^3/s)

	Monthly Average Flows 1957-1984	Monthly Average Flows ± Standard Deviation			Monthly Average Flows 1978	Minimum Daily Flow 1978	Maximum Daily Flow 1978
JAN	2.36	0	to	5.01	1.37	0.62	4.53
FEB	4.64	0	to	9.33	0.50	0.37	0.82
MAR	10.37	5.56	to	15.18	16.28	0.36	79.56
APR	6.53	2.04	to	11.02	7.56	0.48	32.56
MAY	1.99	0	to	3.90	0.73	0.20	1.89
JUN	0.65	0	to	1.91	0.08	0.01	0.20
JUL	0.24	0	to	0.61	0.00	0	0.00
AUG	0.25	0	to	0.67	0.01	0.00	0.04
SEP	0.69	0	to	2.36	0.14	0	0.67
OCT	0.80	0	to	2.51	1.79	0.25	9.77
NOV	2.01	0	to	4.58	0.95	0.23	3.11
DEC	3.93	0.26	to	7.60	2.81	0.40	15.80
ANNUAL MEAN	2.87	1.96	to	3.78	2.71		

Source: Water Survey of Canada 1985.

TABLE E-3

FREQUENCY OF TWENTY MILE CREEK DAILY DISCHARGE IN 1978

Flow (m^3/s)	Frequency[1]
0	0.003
0.003	0.10
0.015	0.20
0.16	0.30
0.35	0.40
0.42	0.50
0.65	0.60
1.16	0.70
1.63	0.80
7.28	0.90
11.81	0.95
57.76	0.99
79.56	1.00

---

[1] Fraction of time that the flow is less than indicated.

Source: Water Survey of Canada 1985.

of Twenty Mile Creek were not available, basic equations of hydraulic geometry were used to estimate the velocity of the creek at its median flow. Hydraulic geometry describes the the effect of a variation in flow on mean depth, width, and velocity at a particular cross-section.

The basic equations of hydraulic geometry are (Park 1977):

$$W = aQ^b$$

$$d = cQ^f$$

$$U = kQ^m$$

Where:

W = width

d = depth

U = velocity

Q = flow

a, b, c, f, k, m = numerical coefficients

In order to better characterize the hydraulic conditions of the creek, a survey was completed on 8 October 1986 (Dillon 1986b). The mean daily flow for that day at the Ball's Falls gauging station was 4.51 m^3/s. Average stream widths varied between 15 m and 30 m with apparent depths of 0.5 m to 1.5 m. Values of Manning's "n" factor were judged to be very high, with an estimated average of 0.05.

The following theoretical average values for the exponents in the basic equations of hydraulics were taken from Park (1977): $b=0.23$, $f=0.42$, and $m=0.35$. For the purposes of the risk assessment, it was assumed that the average width and depth ($d=1$ m and $W=22.5$ m) occurred at the mean flow ($Q=4.51$ m^3/s) observed during the October 1986 survey. Based on this assumption, the other coefficients were calculated as $a=15.91$, $c=0.53$ and $k=0.12$. Therefore, the basic equations of hydraulic geometry for Twenty Mile Creek were expressed as follows:

$$W = 15.91\ Q^{0.23}$$

$$d = 0.53\ Q^{0.42}$$

$$U = 0.12\ Q^{0.35}$$

Given that the median flow over the course of the year was estimated to be 0.42 m^3/s (see Section E.2.2), the average width was calculated as 13.03 m, the average depth as 0.37 m and the average velocity as 0.09 m/s.

E.3 ESTIMATION OF CONTAMINANT CONCENTRATIONS

As discussed in Appendix B, chemicals which spill into water can be classified into three general categories: 1) "mixers", soluble compounds that mix readily with water; 2) "floaters", immiscible (slightly soluble or insoluble) compounds that float on water; and 3) "sinkers", immiscible compounds that sink to the bottom of a watercourse (Thibodeaux 1980). To account for the variability of the composition of the wastes to be transported to the proposed OWMC, one chemical from each of the above categories was selected for analysis: pyridine (the "mixer"); petroleum distillates (the "floater"); and methylene chloride (the "sinker.") The procedure used to select these surrogate chemicals is described in detail in Appendix B.

A series of environmental transport models were used to characterize the behavior of the three types of chemicals in water. These models incorporate the following assumptions:

- The "mixer", pyridine, was assumed to mix rapidly with the stream water. Dilution and dispersion of pyridine in the water were estimated to predict chemical concentrations at various locations downstream from the access road crossing.
- The "sinker", methylene chloride, was assumed to sink and form a separate liquid phase at the bottom of the stream. The rate of dissolution of methylene chloride from this organic phase was estimated to predict the resulting concentrations in water.
- The "floater", petroleum distillates, was assumed to form a separate phase on the water surface. The rate of volatilization of petroleum distillates from Twenty Mile Creek to the atmosphere was used to predict the removal from the stream as the chemical is transported downstream. No mixing into the water was assumed.

Although these models do not consider all the processes that govern the fate and transport of pollutants in the environment, the assumptions made in applying the models are conservative in that they will tend to overstate the extent of contamination under foreseeable circumstances. The models used in the risk assessment are described in this section.

E.3.1 MIXER (PYRIDINE)

E.3.1.1 Theory of the Model

A mass of pyridine was assumed to be spilled instantaneously into Twenty Mile Creek. The distribution of pyridine at various locations following the spill were estimated using the following equation (EPA 1985):

$$C = \frac{(0.001)(M)}{A(4\pi Et)^{1/2}} \exp - \left[\frac{(X - Ut)^2}{4\,Et} + Kt \right]$$

Where:

C = concentration of pyridine, mg/l

M = mass of pyridine spilled, mg

A = cross-sectional area of creek, m^2

X = distance downstream, m

t = time, s

U = average stream velocity, m/s

E = longitudinal dispersion coefficient, m^2/s

K = decay rate constant, s^{-1}

This model incorporates the assumptions that the creek can be represented as an idealized rectangular channel, and that the chemical concentration following a spill is uniform throughout the cross section of the creek.

The maximum concentration at any time, t, after the release occurs at X = Ut. Therefore:

$$C_{max} = \frac{(0.001)(M)}{A\ (4\pi Et)^{1/2}} \exp(-Kt)$$

Based on the model, the concentration distribution along the X-axis (upstream/ downstream axis) would be a symmetrical Gaussian curve centered at the point X = Ut. When concentration is measured as a function of time at a fixed point, X, the distribution is not symmetrical; however, the lack of symmetry becomes small as $Ut \gg (4\ Et)^{1/2}$.

The following relationships were used to derive the parameters required in the application of the model:

- Mass of pyridine spilled

$$M = (V)(\rho)$$

Where:

M = mass of pyridine spilled

V = quantity of pyridine spilled, 227 l, 1 514 l, or 18 925 l

ρ = density of pyridine, 0.983 kg/l

Therefore:

M(227 l) = 223 kg

M(1 514 l) = 1 488 kg

M(18 925 l) = 18 603 kg

- Cross-sectional area of the creek

$$A = Wd$$

Where:

A = cross-sectional area of the creek

W = average width of creek, 13.03 m

d = average depth of creek, 0.37 m

Therefore,

A = 4.8 m^2

- Shear velocity

$$U^* = (gds)^{1/2}$$

Where:

U^* = shear velocity, m/s

g = acceleration due to gravity, 9.8 m/s^2

d = average depth of creek, 0.37 m

s = channel slope 0.0111, 0.0283, or 0.0003 at various segments of the Creek

Therefore,

U^* = 0.201 m/s, 0.321 m/s or 0.034 m/s at various segments of the creek

- The longitudinal dispersion coefficient (Fisher et al. 1979)

$$E = \frac{0.011\ U^2\ W^2}{d\ U^*}$$

Where:

E = longitudinal dispersion coefficient

U = average creek velocity, 0.09 m/s

W = average width of creek, 13.03 m

d = average depth of creek, 0.37 m

U^* = shear velocity, 0.201 m/s, 0.321 m/s, or 0.034 m/s at various segments of the creek

Therefore,

E = 0.20 m^2/s, 0.13 m^2/s, or 1.20 m^2/s at various segments of the creek.

The weighted average value of E for the section of Twenty Mile Creek modeled was used to represent the longitudinal dispersion for the segment to be modeled. This value was calculated as:

E = 0.524 m^2/s

- The decay rate constant
  The decay rate constant is dependent on the overall chemical, physical and biological reactions in the creek. These include hydrolysis, volatilization, photolysis, biodegradation, adsorption, sedimentation, and fish uptake. For this analysis, only volatilization was considered. The volatilization rate was calculated, according to the procedure of Mabey et al. (1982), as 1.54 x 10^{-6} hr^{-1}, or a half-life of 51 years. In comparison to the timescale of the phenomena considered here, this rate was considered to be negligible. Therefore, the decay rate constant (K) was assumed to be 0.

E.3.1.2 Summary of Results

The "mixer" model was used to estimate the concentration of pyridine at various locations downstream (X) and times (t) after each size spill. The maximum concentration values at various points downstream from the Regional Road 24 are presented in Table E-4. At each point X, the maximum concentration occurred at t = X/U. For the 227 liter spill, the maximum concentration calculated 100 meters downstream from the bridge was 541 mg/l; at 3,000 meters, 99 mg/l; and at 6,400 meters, the entrance to Jordan Harbor, 68 mg/l. Similarly, for the 1,514 liter spill, the maximum concentration calculated at a distance 100 meters downstream from the bridge was 3 690 mg/l and at Jordan Harbor, 451 mg/l. For the 18,925 liter spill, TABLE E-4

MAXIMUM CONCENTRATION[1] OF PYRIDINE AT VARIOUS LOCATIONS DOWNSTREAM FROM REGIONAL ROAD 24

Distance Downstream, X (m)	Spill Size (l)		
	227	1 514	18 925
100	541	3 609	45 112
500	242	2 624	20 175
1 000	171	1 141	14 266
1 500	140	932	11 648
2 000	121	807	10 087
2 500	108	722	9 022
3 000	99	659	8 236
3 500	92	610	7 625
4 000	86	571	7 133
4 500	81	538	6 725
5 000	77	510	6 380
5 500	73	487	6 083
6 000	70	466	5 824
6 400	68	451	5 639

---

[1] Concentrations in mg/l

the maximum concentration calculated at a distance 100 meters, was 45 112 mg/l and at Jordan Harbor, 5 639 mg/l.

The concentration distribution along the X-axis at a given time (approximately 6 hours) is presented in Table E-5. The distribution is symmetrical about 2,000 meters and concentrations above 0.01 mg/l are observed from 1,200 to 2,800 meters downstream from Regional Road 24. The concentration distribution at a fixed point, 2,000 meters, at various times is also presented in Table E-5. As shown, it would take approximately 250 minutes, (about four hours) for a plume of pyridine with a concentration greater than 1.0 mg/l to pass a point 2000 meters downstream from Regional Road 24.

E.3.2 SINKER (METHYLENE CHLORIDE)

E.3.2.1 Theory of the Model

Methylene chloride spilled into Twenty Mile Creek was assumed to sink to the bottom and contaminate the immediate vicinity of the spill. As it sinks, the spill of waste would be expected to break up into drops of various sizes. Some of the finest drops would never reach the stream bed, but instead would dissolve and be advected downstream. The majority of the spill was assumed to settle on the stream bed in the shape of a continuous pool, and then slowly dissolve into the water.

TABLE E-5

DISTRIBUTION OF THE CONCENTRATION OF PYRIDINE AT A FIXED TIME AND AT A FIXED LOCATION

At t = 370 min		At X = 2,000 m	
X (m)	C(mg/l)	t(min)	C(mg/l)
1 200	0.01	259	0.19
1 300	0.27	270	9.0
1 400	4	296	150
1 500	50	315	1 130
1 600	325	333	4 100
1 700	1 460	351	8 260
1 800	4 270	370	10 090
1 900	8 140	389	8 020
2 000	10 090	407	4 410
2 100	8 140	426	1 750
2 200	4 270	441	530
2 300	1 460	463	120
2 400	325	481	20
2 500	50	500	4
2 600	4	519	0.47
2 700	0.27	537	0.05
2 800	0.01	556	0.01

---

[1] Spill size: 18 925 l

Two models were used to predict the concentration of methylene chloride just downstream from the pool. The first model (Environment Canada 1985) was used to predict the length and width of the contamination zone on the stream bed. The second model (EPA 1985) was used to predict the dissolution process and the chemical concentration just downstream from the contamination zone. The portion of the chemical that would dissolve immediately before reaching the stream bed was not modeled as it was assumed that it would act as a mixer, and that the concentrations would be lower than those estimated in Section E.3.1. Therefore, the entire quantity of spilled methylene chloride was conservatively assumed to reach the stream bed.

Length of the Contamination Zone. The following equation was used to calculate the length of the contamination zone:

$$X = Ut$$

Where:

X = length of zone of contamination, m

U = average stream velocity, 0.09 m/s (see Section E.2)

t = settling time, s

The following relationships were used to derive the value for settling time (t):

- Minimum drop diameter

$$d_{min} = \frac{\sigma K_F \left[\frac{1000\ D\rho_w}{0.1\ \mu_w} \right]^{3/5}}{10\rho_1 (U_{rel})^{7/5}}$$

Where:

d_{min} = minimum drop diameter, cm

K_F = experimental constant 0.05468

D = puncture size, 0.2 m

ρ_w = density of water, 1.0 g/cm^3

σ = interfacial surface tension, 0.027 N/m

μ_w = viscosity of water, 0.0153 g/(cm-s)

ρ_1 = density of chemical, 1.322 g/cm^3

U_{rel} = relative velocity of chemical, m/s

The following relationships were used to derive the value for relative velocity, U_{rel}:

$$U_{rel} = (U^2 + U^2{}_n)^{1/2}$$

Where:

U_{rel} = relative velocity of chemical, m/s

U = average stream velocity at puncture, 0.09 m/s

U_n = chemical velocity at puncture, m/s

U_n = 0.60 $(2gH)^{1/2}$

Where:

g = acceleration due to gravity, 9.8 m/s^2

H = height of chemical in tank car, 2.75 m

Therefore:

U_n = 4.4 m/s;

U_{rel} = 4.4 m/s; and

d_{min} = 0.0165 cm

- Terminal fall velocity
  Droplets are formed as the chemical discharges from the tank and enters the water. The terminal fall velocity of the droplets in water is dependent on the flow regime of the falling drop. A dimensionless number, K, which represents a ratio of the gravitational to the viscous forces is used to determine into which regime it may be classified:

$$K = d_{min} \left[\frac{g \ \rho_w \ \rho_{DIFF}}{\mu_w^2} \right]^{1/3}$$

Where:

ρ_{DIFF} = density difference between substance and water, 0.322 g/cm^3

g = acceleration due to gravity, 980 cm/s^2

Therefore:

K = 1.82

If the value of K is less than 3.3, the terminal fall velocity is calculated by:

$$U_T = \frac{g \ d^2_{min} \ \rho_{DIFF}}{18 \ \mu_w}$$

Where:

U_T = terminal fall velocity, cm/s

Therefore:

U_T = 0.31 cm/s

The relationship between terminal fall velocity, depth, and settling time is:

$$t = \frac{5}{3} \frac{d}{U_T}$$

Where:

t = settling time, min

U_T = terminal fall velocity, 0.31 cm/s

d = stream depth, 0.37 m

Therefore:

t = 1.98 min.

Based on the calculated value for settling time, the length of the contamination zone (x) was estimated to be 10.7 m.

Width of the Contamination Zone. The following equation was used to estimate the width of the contamination zone:

$$W = \left[\frac{4\ A_R\ d^2}{\pi}\right]^{1/2}$$

Where:

W = width of contamination, m

d = stream depth, 0.37 m (see Section E.2)

A_R = area number (dimensionless)

The following relationships were used to derive the value for the area number (A_R):

- Froude number

$$F_r = \frac{U_n^2}{g\ D}$$

Where:

F_r = Froude number, dimensionless

U_n = chemical velocity at puncture, 4.405 m/s

D = equivalent puncture diameter, 0.2 m

g = acceleration due to gravity, 9.8 m/s^2

Therefore:

F_r = 9.9

- Weber number

$$W_e = \frac{1000\ D\ U_n^2\ \rho_1}{\sigma}$$

Where:

W_e = Weber number, dimensionless

ρ_1 = density of chemical, 1.322 g/cm^3

σ = interfacial surface tension, 0.027 N/m

Therefore:

W_e = 190 015

- Reynolds number

$$R_e = \frac{1000\ D\ U_n\ \rho_1}{\mu_1}$$

Where:

R_e = Reynolds number, dimensionless

μ_1 = chemical viscosity, 0.0004 kg/(m-s)

σ_1 = density of chemical, 1.322 g/cm^3

Therefore:

R_e = 2,911,704

- Height number

H_e = d/D

Where:

H_e = Height number, dimensionless

Therefore:

H_e = 1.85

- Area number

$$A_R = \frac{605\ (F_r)^{0.41}\ (W_e)^{0.61}}{(R_e)^{0.51}\ (H_e)^{1.1}}$$

Where:

A_R = area number, dimensionless

Therefore:

A_R = 660

Based on A_R = 660, the width of the contamination (W) was calculated to be 10.7 m.

Dissolution from the Contamination Zone. The concentration of methylene chloride just downstream from the contamination zone can be estimated using the following expression:

$$C_{WM} = C_L\ \frac{Ws}{W} + C_0\ (1 - \frac{Ws}{W})$$

Where:

C_{WM} = concentration of chemical at complete mixing, mg/l

C_L = concentration of chemical just downstream from the spill zone before complete mixing occurs, mg/l

W_s = spill width, 10.7 m

W = river width, 13.3 m

The value for C_L was calculated using the following equation:

$$C_L = (C_o - C_s) \exp\left[\frac{-D_{cw} L_s}{\delta_d\ Ud}\right] + C_s$$

Where:

C_L = concentration of chemical just downstream from the spill zone, mg/l

C_o = background concentration of chemical, 0 mg/l

C_s = solubility of chemical in water, 16 700 mg/l

D_{cw} = diffusion coefficient of chemical in water, 1.23×10^{-5} cm^2/s

L_s = length of spill zone, 10.7 m

d = stream depth, 0.37 m

U = stream velocity, 9 cm/s

δ_d = thickness of diffusive layer, cm

The following relationship was used to calculate the thickness of the diffusive layer (δ_d):

$$\delta_d = \frac{(11.6)(1.49)\ \upsilon\ (R_h)^{1/6}}{g^{1/2}\ (U)(n)}$$

Where:

δ_d = thickness of diffusive sublayer, m

υ = kinematic viscosity of water, 0.85×10^{-6} m^2/s

R_h = hydraulic radius of the river = Wd/(2d+W), 0.35 m

U = river velocity, 0.09 m/s

n = Manning's coefficient, 0.05

g = acceleration due to gravity, 9.8 m/s^2

Therefore:

δ_d = 5.9×10^{-4} m

Based on the value calculated for δ_d, C_L was calculated to be 11.2 mg/l. Using this value for C_L, the concentration at complete mixing (C_{WM}) was estimated to be 9.6 mg/l. The time required to dissolve the methylene chloride was estimated using the following formula:

$$T_d = \frac{(1\ 000)(M)}{(3\ 600)(C_L)(U)(d)(W_s)}$$

Where:

T_d = time required to dissolve the chemical, hr

M = total amount of pollutant which is dissolved, kg

M = $V\rho$

Where:

V = quantity of methylene chloride spilled, 227 l, 1 514 l, or 18 925 l

ρ = density of methylene chloride 1.322 kg/l.

If we assume that all the amount spilled settles to the bottom, then for the three spill sizes considered,

M (227 l) = 300 kg

M (1 514 l) = 2 002 kg

M (18 925 l) = 25 019 kg

Therefore:

T_d = 21 hours, 143 hours, 1 782 hours

E.3.2.2 Summary of Results

The "sinker" models were used to estimate the concentration of methylene chloride after complete mixing and the time required to dissolve the chemical. For all three spill sizes, the concentration after complete mixing was calculated to be 9.6 mg/l. The time required to dissolve a 227 liter (300 kg), 1 514 liter (2002 kg) and 18 925 liter (25 019 kg) spill was approximately 21 hours, 143 hours, and 1,782 hours, respectively.

E.3.3 FLOATER (PETROLEUM DISTILLATES)

E.3.3.1 Theory of Model

Petroleum distillates, when spilled into Twenty Mile Creek, were assumed to form a pool with a width defined by the confining banks of the stream. The pool will stretch down the stream until it reaches a maximum length, and will then be transported downstream at the velocity of the stream. Volatilization would result in the release of petroleum distillates from the pool as it is transported downstream.

Because of the low solubility of petroleum distillates, it is assumed that no chemical will dissolve into the creek.

- Dimensions of the pool

  To predict the dimensions of the pool of petroleum distillates, the maximum spill radius was estimated using a nomogram (Environment Canada 1985). These radii are a function of spill size and were estimated as 10 m for the 227 l spill; 20 m for the 1 514 l spill; and 50 m for the 18 925 l spill.

  Since the maximum spill radius is greater than half of the stream width, the spill will completely fill the stream. The maximum length therefore is calculated using the following equation:

  $$Lmax = \frac{\pi (Rmax)^2}{W}$$

  Where:

  Lmax = maximum spill length

  Rmax = maximum spill radius, 10 m, 20 m, or 50 m

  W = average stream width, 13.03 m

  Therefore:

  Lmax = 24 m, 96 m or 602 m

- Volatilization rate

  The volatilization rate of petroleum distillates was estimated using the following equation (EPA 1985):

  $$Q = \frac{Kg\ A\ P\ M}{RT}$$

  Where:

  Q = emission rate (g/s)

  Kg = gas mass transfer coefficient (m/s)

  A = area of spill (m^2)

  P = vapor pressure 4 mm Hg

  M = molecular weight 130 g/mol

R = gas constant, 0.0624 (m^3-mm Hg/mol K)

T = temperature, 293 K

and

$$Kg = 0.0048\ U^{0.78}\ d^{-0.11}\ Sc^{-0.67}$$

Where:

U = wind speed 1 m/s

d = diameter of pool = 20m, 40 m or 100 m

Sc = Schmidt number, $Sc^{-0.67} = 0.6$

Therefore:

Kg = 2.07×10^{-3} m/s, 1.92×10^{-3} m/s or 1.74×10^{-3} m/s; and

Q = 18.5 g/s, 68.6 g/s or 387.5 g/s

- Time required for volatilization
The time T_v required to volatilize the chemical is:

$$T_v = \frac{M_v}{Q}$$

Where:

T_v = time required to volatilize the chemical, s

M_v = total amount of chemical which is spilled, g

Q = volatilization rate of chemical, g/s

Therefore:

T_v = 2.9 hr, 5.2 hr or 11.5 hr

- Length of stream affected by the spill
The distance downstream X_v affected by the spill is:

$$X_v = \frac{L_{max}}{2} + UT_v$$

Therefore:

X_v = 951 m, 1,737 m or 4,038 m

E.3.3.2 Summary of Results

The results of the analysis for the floater are presented in Table E-6. As can be seen, the 227 liter spill is volatilized in 2.9 hours, at which time the pool has travelled 951 meters downstream from Regional Road 24. The 1,514 liter spill is volatilized in 5.2 hours, at which time it has travelled 1,737 meters downstream. The 18 925 liter spill is volatilized in 11.5 hours, at which time the pool has travelled 4 038 meters downstream.

TABLE E-6

EMISSION RATE (Q), TIME TO VOLATILIZE (T_V) AND DISTANCE AFFECTED BY PETROLEUM DISTILLATE SPILL (X_V)

	Spill Size (1)		
	227	1 514	18 925
Q (g/s)	18.5	68.4	387.5
Tv (min)	174.0	313.0	692.0
(hr)	2.9	5.2	11.5
Xv (m)	950	1 737	4 040.0

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APPENDIX F

THE ACCUMULATION OF CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS IN BEEF AND MILK

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APPENDIX F

THE ACCUMULATION OF CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS IN BEEF AND MILK

F.1 INTRODUCTION

Polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF) are highly lipophilic, environmentally persistent chemicals. Due to their lipophilic nature, PCDD/PCDF tend to be concentrated and stored in fatty tissues of exposed animals. This phenomenon has raised concerns regarding the potential bioaccumulation of PCDD/PCDF in sources of food, such as beef and milk.

Various laboratory experiments have been conducted to gain a better understanding of the potential for the accumulation of PCDD and PCDF in animals. Studies on the bioaccumulation of 2,3,7,8 tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) in rats have been conducted by Fries and Morrow (1975), Rose et al. (1976), Allen (1975), Kociba et al. (1978) and Van Miller et al. (1976). A study of 2,3,7,8-TCDD uptake by beach mice has also been performed by Thalken and Young (1983). These experiments show that 2,3,7,8-TCDD tends to accumulate primarily in the liver and in fatty tissues. For the rodents studied, levels of 2,3,7,8-TCDD detected in the liver were generally higher than those in fat. However, in an experiment involving laboratory monkeys, the levels of 2,3,7,8-TCDD detected in fat were higher than those measured in the liver (Van Miller et al. 1976).

Data on the bioaccumulation of PCDD and PCDF in livestock, including beef cattle and dairy cows, are limited. Therefore, it is difficult to accurately predict the concentrations of PCDD and PCDF in the tissues of animals exposed to these compounds. In this appendix, studies conducted by Firestone et al. (1979), Parker et al. (1980), Jensen et al. (1981), and Jensen et al. (1982) are reviewed to provide a basis for the estimation of the biotransfer of PCDD and PCDF to beef and milk. Those biotransfer factors are used in Appendix D. The biotransfer of PCDD/PCDF to pork, chicken, and eggs has been estimated using correlations presented by Baes (report in preparation).

If a dairy cow is exposed to feed contaminated with a constant concentration of PCDD/PCDF, levels in milk have been shown to rise to a plateau relatively rapidly. It has been estimated that this plateau, commonly referred to as the steady-state concentration, may be reached within three to four weeks after the cow is initially exposed to the contaminated feed (Fries 1986). Body fat concentrations are expected to rise much more slowly to the steady-state level; the exposure period required to reach steady-state PCDD/PCDF concentrations in adipose tissue has been estimated as approximately six months (Fries 1986). It should be noted that some researchers have concluded that much longer periods of exposure than those estimated by Fries are required to reach steady-state levels (see for example, Jensen 1981).

F.2 THE SINGLE COMPARTMENT ELIMINATION MODEL

Ideally, steady-state measurements would be available for estimating the bioaccumulation of PCDD/PCDF in animal fat and tissue. It is not clear, however, that the experiments conducted to date involve exposures of sufficient duration to permit the direct measurement of steady-state PCDD/PCDF concentrations in either milk or beef. For example, bioaccumulation of 2,3,7,8-TCDD in the body fat of non-lactating cattle has been measured in only one study (Jensen et al. 1981). In the study, heifers were exposed to 2,3,7,8-TCDD for a period of only 28 days, well short of the period believed to be necessary to reach steady-state concentration in body fat.

Given the lack of adequate long-term studies, several researchers have applied a single compartment elimination model to estimate steady-state PCDD/PCDF concentrations in animal fat and tissue. (See, for example, Firestone et al. 1979, and Jensen et al. 1981). The use of this particular model is based, in part, on studies conducted by Piper et al. (1973), Fries and Marrow (1975), and Rose et al. (1976), which indicate that it can adequately describe the dissipation of 2,3,7,8-TCDD in rats.

The single compartment model is based on a relatively simple concept: the rate of accumulation within a compartment is equal to the rate of uptake minus the rate of elimination. If the compartment is defined as an animal, or a part of an animal, this concept can be expressed mathematically as follows:

$$\frac{dC_a}{dt} = k_1C_f - k_2C_a \qquad \text{equation (1)}$$

Where:

C_a = Concentration of contaminant in the animal at time t (ppb)

C_f = Concentration of contaminant in food (ppb)

k_1 = Rate constant for uptake (day^{-1})

k_2 = Rate constant for elimination (day^{-1})

t = time, (day)

Equation (1) can be solved for the contaminant concentration of contaminant in the animal (C_a) at time t:

$$C_a = \frac{k_1}{k_2} C_f[1-\exp(-k_2t)]+C_ao[\exp(-k_2t)] \quad \text{equation (2)}$$

Where:

C_ao = Initial concentration in the animal (ppb)

At steady-state, the contaminant concentration in the animal is constant over time:

$$\frac{dC_a}{dt} = 0 = k_1C_f - k_2C_ass$$

Where:

C_ass = Steady-state concentration in the animal (ppb).

Therefore,

$$C_f = \frac{k_2C_ass}{k_1} \quad \text{equation (3)}$$

By substituting equation (3) into equation (2), the concentration at any time (C_a) can be related to the steady-state value (C_ass) as follows:

$$C_a = C_ass\ [1-\exp\ (-k_2t)] + C_ao\ [\exp\ (-k_2t)]$$

Rearranging for C_ass,

$$C_ass = \frac{C_a - C_ao\ [\exp\ (-k_2t)]}{[1 - \exp\ (-k_2t)]} \quad \text{equation (4)}$$

This equation allows for the calculation of the steady state concentration of a contaminant (C_ass) given the period of exposure (t), the level present at time t (C_a), the initial concentration (C_ao), and the elimination rate constant (k_2). It has been used in this risk assessment as the basis for estimating the steady-state bioconcentration of PCDD/PCDF in milk and beef.

F.3 BIOTRANSFER OF PCDD/PCDF TO MILK

The bioaccumulation of PCDD/PCDF in milk has been examined in two studies: Jensen *et al*. (1982) and Firestone *et al*. (1979). The Jensen study examined only 2,3,7,8-TCDD; Firestone investigated the behavior of various hexa-, hepta-, and octa-chlorinated dibenzo-p-dioxins (HxCDD, HpCDD, and OCDD, respectively).

F.3.1 THE JENSEN ET AL. (1982) STUDY

In the Jensen experiment, three lactating cows were given feed containing 5 parts per trillion (5 ppt) 2,3,7,8-TCDD for 14 consecutive days. The 2,3,7,8-TCDD concentration in the feed was then increased to 15 ppt for the next 14 days. Similarly, increases to 50 ppt, 150 ppt, and finally to 500 ppt were made at the end of 14 day increments. The cows remained on feed containing 500 ppt 2,3,7,8-TCDD for 21 days.

Milk was taken from the cows periodically during the feeding experiment and analyzed for 2,3,7,8-TCDD. The highest 2,3,7,8-TCDD concentration measured in milk from any cow was 89 ppt following 16 days of exposure to feed containing 500 ppt 2,3,7,8-TCDD. It is clear, however, that steady state concentrations of 2,3,7,8-TCDD had not been reached in milk produced by at least two of the three cows when the exposures were terminated after 21 days.

Jensen measured the decline in 2,3,7,8-TCDD concentrations in milk from two of the cows following the completion of the 21 day feeding experiment. Using these dissipation data, Jensen calculated an elimination half life of 41 days for 2,3,7,8-TCDD. The corresponding elimination rate constant (k_2) is equal to 0.693/(41 days), or 0.017 days^{-1}.

The elimination rate constant can be used to estimate a steady-state concentration of 2,3,7,8-TCDD in milk produced by each cow by applying the single compartment model (see equation 4). The calculations are summarized as follows:

Cow	C_m (ppt)	t(d)	$C_m o$ (ppt)	k_2 (d^{-1})	$C_m ss$ (ppt)
A	79	21	16	0.017	226
B	89	16	19	0.017	313
C	68	21	14	0.017	194

Where:

C_m = Observed 2,3,7,8-TCDD concentration (ppt) in milk at time t;

C_mo = 2,3,7,8-TCDD concentration in milk (ppt) at the start of the 21 day feeding experiment.

C_mss = Calculated steady-state 2,3,7,8-TCDD concentration in milk (ppt).

Bioconcentration factors (BCF) for 2,3,7,8-TCDD in the milk from each cow can be calculated by dividing the steady-state concentrations (C_{mss}) by the feed concentration (C_f). A similar parameter, the biotransfer factor (BTF), as defined by Travis et al. (1987), is used to evaluate bioaccumulation in this risk assessment (see Appendix D). The biotransfer factor relates the concentration in milk (or tissue) to the level of chemical exposure as follows:

$$\text{BTF} = \frac{\left[\dfrac{\text{mg contaminant in milk (or tissue)}}{\text{kg milk (or tissue)}}\right]}{\left[\dfrac{\text{mg contaminant in feed}}{\text{kg feed}} \times \dfrac{\text{kg feed}}{\text{day}}\right]}$$

The BTF can be calculated by dividing the BCF by the feed rate, 16 kg/d in this experiment.

Cow	C_mss(ppt)	C_f(ppt)	BCF, milk (mg/kg)/(mg/kg)	BTF, milk (mg/kg)/(mg/d)
A	226	500	0.45	0.028
B	313	500	0.63	0.039
C	194	500	0.39	0.024

The biotransfer factor for 2,3,7,8-TCDD to milk can be estimated as the average of the BTF values calculated for the individual cows, or about 0.03 (mg/kg)/(mg/d).

F.3.2 THE FIRESTONE ET AL. (1979) STUDY

The BTF values for the more highly chlorinated forms (congeners) of PCDD can be derived from data collected by Firestone et al. (1979). In this experiment, three dairy cows were administered technical grade pentachlorophenol (tPCP) contaminated with detectable levels of several chlorinated dioxins. The concentrations of PCDD in the feed can be calculated as follows:

$$C_f = \frac{(D_p)(C_p)(W)}{(10^6)(F)}$$

Where:

C_f = PCDD concentration in feed, (ppb)

D_p = Dose of tPCP to the cow, 10 mg/(kg-d)

C_p = PCDD concentration in tPCP, (ppm)

W = average weight of the cows tested, 599 kg

F = assumed feed rate, 16 kg/d (Actual feed rate not reported.)

The PCDD concentrations in the feed are summarized below:

Congener*	Concentration in tPCP(ppm)	Concentration in feed (ppb)
1,2,4,6,8,9-HxCDD	2.0	0.75
1,2,3,6,8,9-HxCDD	7.8	2.9
1,2,3,6,7,8-HxCDD	10	3.7
1,2,3,4,6,7,9-HpCDD	75	28
1,2,3,4,6,7,8-HpCDD	205	77
OCDD	690	260

* The numerical prefix indicates the location of the chlorine atoms on the PCDD/PCDF molecule. See Appendix C for a detailed explanation of the numbering system for identifying the individual congeners of PCDD/PCDF.

Several additional PCDD congeners were detected at levels less than 1 ppm. No polychlorodibenzofurans (PCDF) were reported.

Three PCDD congeners (1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD, and OCDD) were detected in the milk fat collected and analyzed during the experiment. Although the cows were administered tPCP for a full 70 days, steady-state levels of these congeners in milk do not appear to have been reached in the Firestone et al. experiment. Based on a dissipation study, however, Firestone et al. calculated elimination rate constants (k_2) for each congener detected in the milk. The single compartment model (see equation 4) can therefore be used to estimate steady-state concentrations in body fat ($C_{fat}ss$):

Congener	C_{fat} (ppb)	t(d)	$k_2(d^{-1})$	$C_{fat}ss$ (ppb)*
1,2,3,6,7,8-HxCDD	19	70	0.0137	31
1,2,3,4,6,7,8-HpCDD	39	70	0.0147	61
OCDD	24	70	0.0168	35

* Since data on PCDD concentrations in milk fat at the beginning of the experiment are not available, these initial concentrations ($C_{fat}o$) were assumed to be zero.

It is interesting to note that the elimination rate constants derived by Firestone for the HxCDD, HpCCD, and OCDD cogeners are very similar in magnitude to that estimated for 2,3,7,8-TCDD by Jensen et al. (0.017 days^{-1}). It should also be recognized that, only those congeners with chlorine atoms in the 2,3,7, and 8 positions (the "lateral" positions) of the PCDD molecule were detected in the milk samples. According to Firestone, "This suggests that dioxin congeners with low or zero biological activity (lateral ring positions incompletely filled with halogen atoms) are rapidly metabolized or excreted, whereas the biologically active congeners with halogen in all lateral ring positions are relatively stable after absorption by the animal." These findings and conclusion are in agreement with many reports that non-2,3,7,8 substituted PCDDs accumulate in fat to a much lesser extent than those that are laterally substituted, i.e., substituted in the 2,3,7, and 8 positions (Rappe et al. 1984).

If it is assumed that the milk taken from the cows contains 4% fat (Hodgman et al. 1954), corresponding steady-state PCDD concentrations in milk can be estimated:

Congener	$C_{fat}ss$ (ppb)	C_mss (ppb)
1,2,3,6,7,8-HxCDD	31	1.2
1,2,4,6,7,8-HpCDD	61	2.4
OCDD	35	1.4

Bioconcentration factors (BCF) can then be calculated by dividing the steady-state milk values (C_mss) for each congener by the concentrations administered with the feed

(C_f). Corresponding biotransfer factors (BTF) can be determined by dividing the BCF values by an assumed feed rate of 16 kg/d.

Congener	$C_m$ss (ppb)	C_f (ppb)	BCF, milk (mg/kg)/(mg/kg)	BTF, milk (mg/kg)/(mg/d)
1,2,3,6,7,8-HxCDD	1.2	3.7	0.32	0.020
1,2,4,6,7,8-HpCDD	2.4	77	0.031	0.002
OCDD	1.4	260	0.0054	0.0003

F.3.3 SUMMARY OF RESULTS

The Jensen et al. (1982), and Firestone et al. (1979) experiments provide a basis for estimating milk BTFs for tetra-, hexa-, hepta-, and octa-chlorinated dibenzo-p-dioxins. No data, however, are currently available with respect to the accumulation of pentachlorodibenzo-p-dioxins (PnCDD) or any of the polychlorodibenzofurans (PCDFs) in milk. To extend the analysis to these compounds, the following assumptions have been made:

- A BTF midway between the values calculated for laterally substituted TCDD and HxCDD, can be assigned to 2,3,7,8-PnCDDs.
- PCDFs have the same BTF values as the corresponding PCDDs. This assumption is based on the structural similarities between the two groups of compounds.

Using these assumptions, the results of the analysis can be summarized as follows:

Congeners	BTF, milk (mg/kg)/(mg/d)
2,3,7,8-TCDD/TCDF	0.03
2,3,7,8-PnCDDs/PnCDFs	0.025
2,3,7,8-HxCDDs/HxCDFs	0.02
2,3,7,8-HpCDDs/HDCDFs	0.002
OCDD/OCDF	0.0003

F.4 BIOTRANSFER OF PCDD/PCDF TO CATTLE BODY FAT

Three studies have examined the bioaccumulation of PCDD/PCDF in the body fat of cattle: Firestone et al. (1979); Parker et al. (1980); and Jensen et al. (1981). These three investigations are summarized below:

Study	Type of Animal	PCDD/PCDF Considered
Firestone et al. (1979)	Lactating dairy cows	HxCDD, HpCDD, OCDD
Parker et al. (1980)	Non-lactating heifers	HxCDD, HpCDD, OCDD HpCDF, OCDF
Jensen et al. (1981)	Non-lactating heifers	2,3,7,8-TCDD

The Firestone et al. study also examined the accumulation of PCDD in milk and was introduced in the preceding section of this appendix.

F.4.1 THE FIRESTONE ET AL. (1979) STUDY

In the Firestone study, three dairy cows were exposed to PCDD-contaminated pentachlorophenol (tPCP) for a period of 70 days. PCDD measurements in both milk fat and body fat were taken on the 69th day of exposure. Average PCDD levels in milk fat and body fat were measured again 100 days after the administration of PCDD-contaminated PCP had ceased. The results of these analyses are presented below:

	Concentration, (ppb) Day 69			Concentration, (ppb) Day 69+100		
	HxCDD	HpCDD	OCDD	HxCDD	HpCDD	OCDD
Milk Fat	19	39	24	4.3	6.9	3.0
Body Fat	13	24	32	2.5	6.6	5.6

According to Firestone, "These results indicate that the dioxin content in the composite milk fat is similar to that in body fat." On this basis, it may be appropriate to assume that, for lactating cows, the biotransfer factor for PCDD to body fat is approximately the same as that for PCDD to milk fat. It is possible, however, that there may be differences between the partitioning of PCDD within lactating and non-lactating animals (Fries 1986).

F.4.2 THE JENSEN ET AL. (1981) STUDY

The Jensen experiment consisted of two parts: (1) a study of the tissue distribution of 2,3,7,8-TCDD, and (2) a study of 2,3,7,8-TCDD dissipation from fat. Seven heifers were fed rations contaminated with 24 ppt of 2,3,7,8-TCDD for a period of 28 days. The distribution of 2,3,7,8-TCDD within various tissues was studied by sacrificing three of the treated heifers and analyzing samples of muscle, fat, liver and kidney. Dissipation of 2,3,7,8-TCDD in body fat was measured by analyzing samples taken by biopsy from the remaining four calves at intervals after discontinuance of 2,3,7,8-TCDD in the diet.

At the end of the 28 day feeding period, concentrations of 2,3,7,8-TCDD in the body fat of the heifers ranged from 66 ppt to 100 ppt, with an average of approximately 85 ppt. These concentrations cannot be considered steady-state, however, due to the short period of exposure. Jensen therefore used data from the dissipation study to calculate an elimination half-life of approximately 116 days, corresponding to a 2,3,7,8-TCDD elimination rate constant (k_2) of 0.006 d^{-1}. This value can be used in the single compartment model to estimate a steady-state 2,3,7,8-TCDD concentration of approximately 550 ppt in body fat, a value 6 to 7 times higher than detected after 28 days. (It should be noted that Jensen et al. apparently used a different weighting scheme in their analysis, and estimated the steady-state 2,3,7,8-TCDD residue level to be 594 ppt.) As stated by Jensen et al., the steady-state concentration estimated for the body fat in the heifers ". . . is close to the 540 ppt found in the fat of rats fed 22 ppt of TCDD for two years (Kociba et al. 1978)." Therefore, the extent of 2,3,7,8-TCDD bioaccumulation in the fat of cattle appears to be similar to that in the adipose tissue of rats.

The bioconcentration factor (BCF) in beef fat can be calculated by dividing the steady-state fat concentrations ($C_f$ss) by the concentration in the feed (C_f). The biotransfer factor (BTF) to beef fat can then be determined by dividing the BCF by the feed rate of 6 kg/d for the heifers. Finally, the BTF for beef can be calculated by assuming a fat content of 20 percent in meat consumed by humans (Watt and Merrill 1975).

	$C_f$ss(ppt)	C_f(ppt)	BCF, fat (mg/kg)/(mg/kg)	BTF (mg/kg)/(mg/d) fat	beef
2,3,7,8-TCDD	550	24	22.9	3.8	0.8

F.4.3 THE PARKER ET AL. (1980) STUDY

The study conducted by Parker *et al*. involved the feeding of PCDD/PCDF-contaminated PCP to three groups of three heifers. The groups were administered different doses of PCDD/PCDF; the total period of exposure for each group was 160 days. At the end of the 160 day period, the animals were sacrificed, and levels of PCDD/PCDF in blood, liver, and body fat were measured. Unlike the Jensen study, the dissipation of PCDD/PCDF from fat was not examined.

The heifers in the study were fed mixtures of two grades of PCP: technical (tPCP) and analytical (aPCP). The PCDD/PCDF tentatively identified in the aPCP and tPCP at concentrations greater than 5 ppm are presented below:

Congener (Identification Tentative)	Concentration (ppm) aPCP	tPCP
1,2,3,7,8,9-HxCDD, or	<1	5
1,2,3,6,7,8-HxCDD	<1	10
1,2,3,4,6,7,9-HpCDD	<10	132
1,2,3,4,6,7,8-HpCDD	<10	278
OCDD	<20	1 500
1,2,3,4,6,8-HxCDF, or	<5	16
1,2,4,6,7,8-HxCDF, or	<5	28
1,2,4,6,8,9-HxCDF	<5	13
1,2,3,4,6,7,8-HpCDF	<5	28
1,2,3,4,6,8,9-HpCDF	<5	99
OCDF	<10	90

As shown, the concentrations of PCDD/PCDF in aPCP could not be quantified in the experiment, but are considerably lower than those detected in the tPCP.

The three groups of heifers were exposed to different mixtures of aPCP and tPCP. The PCP administered to Group A was 10% tPCP/90% aPCP; Group B, 35% tPCP/65% aPCP; Group C, 100% tPCP. The total dose of PCP to each group remained constant at 15 mg/(kg-d) for the final 118 days of the feeding experiment. The weight and feed rate for the heifers were not specified in the experiment; therefore a body weight of 300 kg and a feed rate of 6 kg/d were assumed. Using these assumptions, the concentration of each PCDD/PCDF congener in the feed for the three groups of animals can be estimated:

Congener	Feed Concentration (ppb)* A (10% tPCP)	 B (35% tPCP	 C (100% tPCP)
1,2,3,7,8,9-HxCDD	0.38	1.3	3.8
1,2,3,6,7,8-HxCDD	0.75	2.6	7.5
1,2,3,4,6,7,9-HpCDD	9.9	35	99
1,2,3,4,6,7,8-HpCDD	21	73	210
OCDD	113	395	1 130
1,2,3,4,6,8-HxCDF	1.2	4.2	12
1,2,4,6,7,8-HxCDF	2.1	7.4	21
1,2,4,6,8,9-HxCDF	0.98	3.4	9.8
1,2,3,4,6,7,8-HpCDF	2.1	7.4	21
1,2,3,4,6,8,9-HpCDF	7.4	26	74
OCDF	6.8	24	68

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* PCDD/PCDF concentrations in aPCP were assumed to be negligible. If actual levels in aPCP are close to the detection limits, some BTF values may be over-estimated.

The levels of the PCDD/PCDF detected in the adipose tissue of the heifers following the 160 day feeding experiment are summarized below:

Congener	Fat Concentration (ppb) A (10% tPCP)	 B (35% tPCP)	 C (100% tPCP)
1,2,3,7,8,9-HxCDD	ND	ND	0.7
1,2,3,6,7,8-HxCDD	1	10	16
1,2,3,4,6,7,8-HpCDD	7	12	52
OCDD	13	36	61
1,2,3,4,6,7,8-HpCDF	0.5	4.2	6.9
OCDF	4	5	7

As in the experiment conducted by Firestone et al. (1979) with milk cows, only those PCDD and PCDF chlorinated in the lateral (2,3,7,8) positions were found to accumulate in fat. This phenomenon is attributed by Parker et al. "... to the fact that an open lateral position provides an opportunity for the metabolism of dioxins and furans to more polar compounds which can be excreted."

Although the Parker study involved a relatively long exposure period (160 days), no evidence is presented in the report to demonstrate that steady-state PCDD/PCDF concentrations were reached. Since an examination of the dissipation of PCDD/PCDF in the fat was not conducted by Parker et al., an elimination half-life for the various congeners cannot be estimated from data generated in the experiment. It was necessary, therefore, to approximate an elimination half-life by reference to the results of the other studies reviewed.

The studies conducted by Jensen et al. (1982) and Firestone et al. (1979) using dairy cows, found half-lives for laterally substituted TCDD, HxCDD, HpCDD and OCDD in milk fat to range from 41 days for TCDD and OCDD to 51 days for HxCDD. If it is likewise assumed that the half-lives for laterally substituted PCDDs and PCDFs in the adipose tissue of non-lactating cattle are similar, then the half-life value of 116 days estimated by Jensen et al. (1981) for 2,3,7,8-TCDD can be applied to other 2,3,7,8-substituted PCDD congeners as well. This allows the use of the single compartment model to calculate steady-state concentrations for the PCDD congeners studied by Parker et al.

The elimination rate constant corresponding to a half-life of 116 days is $0.006\ d^{-1}$. Using this value, the following steady-state PCDD/PCDF concentrations in adipose tissue can be calculated:

Congener	Steady-state Fat Concentration (ppb) A (10% tPCP)	B (35% tPCP)	C (100% tPCP)
1,2,3,7,8,9-HxCDD	ND	ND	1.1
1,2,3,6,7,8-HxCDD	1.6	16	26
1,2,3,4,6,7,8-HpCDD	11	19	84
OCDD	21	58	99
1,2,3,4,6,7,8-HpCDF	0.8	6.8	11
OCDF	6.5	8.1	11

Dividing the steady-state fat concentrations by the feed concentrations yields bioconcentration factors (BCF):

Congener	BCF (mg/kg)/(mg/kg) A (10% tPCP)	B (35% tPCP)	C (100% tPCP)
1,2,3,7,8,9-HxCDD	-	-	0.3
1,2,3,6,7,8-HxCDD	2.1	6.1	3.5
1,2,3,4,6,7,8-HpCDD	0.52	0.26	0.41
OCDD	0.19	0.15	0.088
1,2,3,4,6,7,8-HpCDF	0.38	0.92	0.52
OCDF	0.95	0.34	0.16

The bioconcentration factor for each PCDD/PCDF congener can be estimated by averaging the values corresponding to each group of heifers:

Congener	BCF (mg/kg)/(mg/kg)
1,2,3,7,8,9-HxCDD	0.3
1,2,3,6,7,8-HxCDD	3.9
1,2,3,4,6,7,8-HpCDD	0.40
OCDD	0.14
1,2,3,4,6,7,8-HpCDF	0.61
OCDF	0.48

The low BCF value for 1,2,3,7,8,9-HxCDD is questionable for the following reasons: 1) it is based on only one data point, 2) this congener was not detected in liver samples, which had much higher levels than the fat for all other forms of PCDD/PCDF, and 3) the trend observed in the analysis of the biotransfer of PCDD/PCDF to milk and to beef is for decreasing accumulation with increasing degree of chlorination. Accordingly, the BCF value for 1,2,3,6,7,8-HxCDD has been used to represent the HxCDD congeners.

Biotransfer factors to beef for the various PCDD/PCDF congeners examined in the Parker study can be calculated by dividing the appropriate BCF values by the assumed feed rate for heifers (6 kg/d), and multiplying by the approximate fat content in beef (20%):

Isomer	BCF, fat (mg/kg)/(mg/kg)	BTF, beef (mg/kg)/(mg/d)
HxCDD	3.9	0.13
HpCDD	0.40	0.013
OCDD	0.14	0.005
HpCDF	0.61	0.020
OCDF	0.48	0.016

F.4.4 SUMMARY OF RESULTS

The Jensen *et al*. (1981) and Parker *et al*. (1980) studies provide data for estimating the beef BTFs for tetra-, hexa-, hepta-, and octa-chlorinated dibenzo-p-dioxins, and for hepta- and octa- chlorinated dibenzofurans. The data on the PCDFs are limited, however; and no information with regard to the accumulation of pentachlorinated dibenzo-p-dioxins (PnCDD) are currently available. To allow for a consideration of these compounds in the risk assessment, the following assumptions have been made:

- The BTF for 2,3,7,8-PnCDDs lies midway between the values for 2,3,7,8-TCDD and 2,3,7,8-HxCDDs.
- Corresponding PCDDs and PCDFs have the same BTF values. Therefore, the BTF values for HpCDD and HpCDF, and for OCDD and OCDF, have been averaged. Other PCDF BTF values are assumed to be equal to those of the corresponding PCDD. These assumptions are based on the structural similarities between the two groups of compounds.

Using these assumptions, the results of the analysis can be summarized as follows:

Isomers	BTF, beef (mg/kg)/(mg/d)
2,3,7,8-TCDD/TCDF	0.8
2,3,7,8-PnCDDs/PnCDFs	0.47
2,3,7,8-HxCDDs/HxCDFs	0.13
2,3,7,8-HpCDDs/HpCDFs	0.017
OCDD/OCDF	0.010

F.5 CONCLUSIONS

The data for estimating PCDD/PCDF biotransfer to milk and beef are limited. However, two general observations can be made:

1. The extent of PCDD/PCDF accumulation in fat appears to be inversely related to the extent of chlorination.
2. Those PCDD/PCDF chlorinated in the lateral (2,3,7,8) positions appear to accumulate to a much greater extent than those that are not laterally substituted. This conclusion is well supported by other data from animals (Rappe _et_ _al_. 1984) and humans (Ryan 1986; Rappe _et_ _al_. 1986).

Based on the available data, the following biotransfer factors (BTF) have been estimated for the 2,3,7,8-substituted PCDD/PCDF:

Congener*	BTF (mg/kg)/mg/d Milk	Beef
2,3,7,8-TCDD/TCDF	0.03	0.8
2,3,7,8-PnCDDs/PnCDFs	0.025	0.47
2,3,7,8-HxCDDs/HxCDFs	0.020	0.13
2,3,7,8-HpCDDs/HpCDFs	0.002	0.017
OCDD/OCDF	0.0003	0.010

* The BTF values for non-2,3,7,8-PCDD/PCDF are assumed to be negligible.

In the Phase 4B risk assessment, PCDD/PCDF emissions are expressed in "2,3,7,8-TCDD equivalents" (TEQ). (The TEQ concept is discussed in detail in Appendix H.) It is therefore necessary to calculate a weighted-average BTF based on the estimated emission rates for individual PCDD/PCDF congeners. This can be accomplished using the following equation:

$$BTF_a = \frac{1}{E_t}\sum^{n} (E_i)(BTF_i) \qquad \text{(equation 5)}$$

Where:

BTF_a = Weighted-average-biotransfer factor, (mg/kg)/(mg/d)

BTF_i = Biotransfer factor for an individual congener of PCDD/PCDF, (mg/kg)/(mg/d)

E_t = Total TEQ emission rate (g/s)

E_i = TEQ emission rate for an individual congener of PCDD/PCDF (g/s)

n = The number of congeners considered.

In Appendix H, TEQ emission rates based on schemes developed by the United States Environmental Protection Agency (USEPA) and the Ontario Ministry of the Environment (OMOE) were estimated for the various groups of PCDD/PCDF. The TEQ emission rates calculated using the USEPA scheme are presented in Table F-1 along with the estimated biotransfer factors. Application of equation (5) yields a weighted-average BTF of approximately 0.02 (mg/kg)/(mg/d) for milk and 0.3 (mg/kg)/(mg/d) for beef. The weighted-average BTF values calculated using TEQ emission rates based on the OMOE methodology are presented in Table F-2. These values, 0.004 (mg/kg)/(mg/d) for milk and 0.05 (mg/kg)/(mg/d) for beef, are lower than those calculated using the USEPA scheme. This is due to the fact that the OMOE TEQ emissions are more heavily composed of the highly chlorinated congeners that accumulate to a lesser extent than lower chlorinated congeners.

It should be recognized that there is considerable uncertainty associated with these estimates due to limitations in the data available with respect to the bioaccumulation of

TABLE F-1

ESTIMATED USEPA TEQ EMISSIONS AND BTF FOR POLYCHLORINATED DIBENZO-P--DIOXINS (PCDD) AND POLYCHLORINATED DIBENZOFURANS (PCDF)

Congener Group	TEQ Emission[1] Rates (g/s)	BTF Factor (mg/kg)/(mg/d) Milk	Beef
PCDD			
Tetra			
2378	2.3 E-10	0.03	0.8
non 2378	4.1 E-11	0	0
Penta			
2378	3.0 E-10	0.025	0.47
non 2378	2.7 E-11	0	0
Hexa	4.5 E-10	0.02	0.13
Hepta	1.3 E-11	0.002	0.017
Octa	0.0 E+00	0.0003	0.010
PCDF			
Tetra			
2378	2.8 E-10	0.03	0.8
non 2378	3.3 E-11	0	0
Penta			
2378	6.4 E-10	0.025	0.47
non 2378	6.1 E-11	0	0
Hexa	9.9 E-10	0.02	0.13
Hepta	7.3 E-11	0.002	0.017
Octa	0.0 E+00	0.0003	0.010
Weighted Average		0.02	0.3

---

[1] Based on the USEPA toxic equivalent Scheme (see Table H-3, Appendix H)

TABLE F-2

ESTIMATED OMOE TEQ EMISSIONS AND BTF FOR POLYCHLORINATED DIBENZO-P-DIOXINS (PCDD) AND POLYCHLORINATED DIBENZOFURANS (PCDF)

Congener Group	TEQ Emission[1] Rates (g/s)	BTF Factor (mg/kg)/(mg/d) Milk	BTF Factor (mg/kg)/(mg/d) Beef
PCDD			
Tetra			
2378	2.3 E-10	0.03	0.8
non 2378	4.1 E-11	0	0
Penta			
2378	6.1 E-11	0.025	0.47
non 2378	5.4 E-10	0	0
Hexa	1.1 E-09	0.02	0.13
Hepta	1.3 E-10	0.002	0.017
Octa	3.7 E-12	0.0003	0.010
PCDF			
Tetra			
2378	1.4 E-09	0.03	0.8
non 2378	1.6 E-08	0	0
Penta			
2378	3.2 E-09	0.025	0.47
non 2378	3.1 E-08	0	0
Hexa	9.9 E-09	0.02	0.13
Hepta	7.3 E-10	0.002	0.017
Octa	4.5 E-12	0.0003	0.010
Weighted Average		0.004	0.05

---

[1] Based on the OMOE toxic equivalent Scheme (see Table H-3, Appendix H)

PCDD/PCDF in milk and beef. Furthermore, experiments conducted to date have been of too short duration to provide direct measurements of the steady-state concentrations of PCDD and PCDF and the accuracy of the single compartment elimination model in predicting these concentrations has not been established. It is believed, however, that the approach described in this appendix provides conservative, yet reasonable, estimates of the biotransfer of PCDD/PCDF to milk and beef.

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APPENDIX G

USE OF A THRESHOLD MODEL FOR THE ESTIMATION OF RISK ASSOCIATED WITH EXPOSURE TO CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS

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APPENDIX G

USE OF A THRESHOLD MODEL FOR THE ESTIMATION OF RISK ASSOCIATED WITH EXPOSURE TO CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS

G.1 INTRODUCTION

As discussed in Chapter 6 of this report, the assessment of risk associated with exposure to carcinogens differs from that for non-carcinogens. For non-carcinogens, it is believed that a specific dose must be exceeded before an irreversible toxic effect is produced. The exposure level above which effects may be possible is referred to as the threshold dose. For carcinogens, however, it is usually assumed that no such threshold dose exists, i.e. that some risk is associated with any level of exposure and that risk increases with increasing exposure. Therefore, no-threshold models have been developed to estimate the incremental cancer risk associated with exposure to low doses of carcinogens.

2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) has been identified as an extremely potent carcinogen in laboratory animals (CAG 1985). In the Phase 4A Risk Assessment, ENVIRON estimated the risks associated with exposure to all suspected carcinogens, including 2,3,7,8-TCDD, using a no-threshold model. This approach is consistent with the methodology currently applied by the United States Environmental Protection Agency (EPA) in performing carcinogen risk assessments. However, several governments and regulatory bodies, including the Ontario Ministry of the Environment (MOE), have concluded that a threshold model is more appropriate in assessing cancer risks for 2,3,7,8-TCDD. This conclusion is based on beliefs regarding the general mechanism by which 2,3,7,8-TCDD is thought to cause cancer.

The use of a no-threshold model in evaluating the risks for carcinogens stems from our knowledge of and experience with radiation. It has been shown that radiation causes both mutations and cancers at exposure levels that cause no immediate, overt effects and that there are no discernible thresholds for radiation-induced mutagenicity and carcinogenicity. This finding contributed significantly to the general conclusion that a threshold dose does not exist for many carcinogens. Despite this general conclusion, certain carcinogens do not currently appear to meet the criterion for a no-threshold model because they are not known to be mutagens. In particular, 2,3,7,8-TCDD does not appear to be a mutagen; therefore, there is a basis for considering a threshold model in evaluating the toxicity of 2,3,7,8-TCDD.

A threshold model embodies the assumption that there is no risk associated with doses below a certain critical level. This is the procedure normally used in establishing safe levels of exposure to noncarcinogens. In establishing such a safe level, a dose at which no toxicity is observed, the NOAEL (no-observed-adverse-effect level), is usually determined in animal experiments. That dose is divided by 10 to allow for the possibility that humans are more sensitive to the chemical than the laboratory animals and by another factor of 10 to allow for variations in sensitivities among humans. The dose calculated by dividing the NOAEL by 100 is then set as an acceptable exposure level. Some experiments do not permit determination of a NOAEL because there is some toxicity at the lowest tested dose. Such experiments do, however, permit the setting of a LOAEL (lowest-observed-adverse-effect-level). When used to set human exposure levels, LOAELs are divided by factors of 100 to 1,000 depending upon the severity of the toxic effects.

The choice between a no-threshold and a threshold model for any carcinogen may have a significant effect on the level of exposure that is considered acceptable. With any no-threshold model, some finite risk is associated with all exposures greater than zero. Safe doses calculated using a threshold model for 2,3,7,8-TCDD are typically one order of magnitude greater than the dose corresponding to a 1 x 10^{-6}

cancer risk using a no-threshold model. Therefore, the use of a no-threshold model is more conservative (i.e., less likely to underestimate actual risk) than the use of a threshold model.

G.1.1 2,3,7,8-TCDD AND THE MECHANISM OF CARCINOGENESIS

Carcinogenesis is generally believed to be at least a two-stage process. The first step in carcinogenesis, "initiation", is widely believed to involve a heritable alteration in DNA structure, i.e., a "mutation-like" event. This hypothesis is consistent with observations that all known initiators are capable of causing DNA damage and that the initiation step is relatively irreversible (Farber and Cameron 1980; Slaga 1983). The second step in carcinogenesis is known as "promotion". Although the nature of promotion is not clear, it is generally believed to require continual exposure to the promoting agent during the prolonged period of tumor development. Unlike initiation, promotion is believed to be a reversible process.

Many carcinogens are considered to be complete, that is, capable of both initiating and promoting tumor development. However, it has been postulated that some chemicals that increase the incidence of tumors in experimental cancer bioassays do not interact with DNA and cause mutations, i.e., they are not initiators. These carcinogens, termed promoters, increase the incidence of tumors only when exposure is prolonged (as in a cancer bioassay) and when the cells that ultimately develop into tumors have previously been initiated (as may result from naturally-occurring initiators, such as nitrosamines in food, or background radiation).

Although details remain uncertain, the mechanism of promotion can be pictured as a series of toxic insults to an initiated cell. If exposure to a promoter ceases before a sufficient number of insults occurs, the cell can repair, or survive with, the damage. However, if exposure to the promoter continues at a sufficient level, a threshold is crossed, the damage inflicted is irreparable, and the cell will become malignant. This general understanding of the mechanism of promotion suggests that it may be appropriate to evaluate the risks associated with a promoter using a threshold model.

Research performed to date indicates that 2,3,7,8-TCDD may not be an initiator. Specifically, most evidence supports the conclusion that 2,3,7,8-TCDD neither binds to DNA (Poland and Glover 1979) nor causes mutations (Nebert et al. 1976; McCann, cited in Wassom 1977, Geiger and Neal 1981; Gilbert et al. 1981; Mortelmans et al. 1984). More directly, 2,3,7,8 has been observed to act as a promoter, not an initiator, in two initiation-promotion tests. (Poland et al. 1982; Pitot et al. 1980). This evidence supports the conclusion that 2,3,7,8 TCDD does not cause the irreversible initiation step in carcinogenesis.

G.1.2 CALCULATION OF AN ACCEPTABLE DOSE FOR 2,3,7,8-TCDD

Based on the belief that 2,3,7,8-TCDD acts as a promoter and has a threshold for activity, the Ontario Ministry of the Environment (OMOE) has developed an acceptable daily intake (ADI) value for 2,3,7,8-TCDD. This ADI value has been derived using data from studies conducted by Kociba et al. (1978) and Murray et al. (1979). The use of these studies by OMOE in calculating the ADI for 2,3,7,8-TCDD is summarized below.

Based on a study of tumors in rats, a NOAEL of 1 ng/kg/d* has been estimated for 2,3,7,8-TCDD (Kociba et al. 1978). In a separate study involving laboratory mice, Murray et al. (1979) concluded that a NOAEL of 1 ng/kg/d was also demonstrated for reproductive effects. These NOAEL values were both determined in experiments involving lifetime exposure and an appropriate safety factor was determined to be 100 (10 for possible variation between rat and man and an additional factor of 10 for interhuman variation) (NRC 1977). Therefore, an acceptable level for lifetime daily intake for 2,3,7,8-TCDD was calculated as follows:

$$\text{ADI} = \frac{\text{NOAEL}}{\text{Safety Factor}} = 10 \text{ pg/kg/d} \,^{**}$$

---

* ng/kg/d: nanograms (10^{-9} g) per kilogram body weight per day

** pg/kg/d: picograms (10^{-12} g) per kilogram body weight per day

Where:

NOAEL = 1.0 ng/kg/d

Safety Factor = 100

Several governments have also calculated an ADI for 2,3,7,8-TCDD. These countries, listed in Table G-1, base their actions on the NOAEL from chronic bioassays performed by Kociba et al. (1978). The safety factor selected by some countries is higher than 100-fold, which is the standard safety factor for data from a chronic animal study. The reason for the higher safety factors may involve the fact that it is unusual to apply a safety factor to a threshold for a carcinogen; furthermore, the potency of 2,3,7,8-TCDD may call forth greater concern than other chemicals.

G.2 EXAMINATION OF POSSIBLE MECHANISM(s) OF CARCINOGENICITY

Any attempt to explain the mechanism of cancer development must be able to account for a variety of observations relating to the phenomenon of cancer. A large body of information implicates alteration of cellular DNA in the process of carcinogenesis. Some of the evidence for this includes:

- Many (though not all) chemical carcinogens either are electrophilic reactants, or are metabolized to electrophilic reactants, which are capable of binding

TABLE G-1

ACCEPTABLE DAILY INTAKE (ADI) LEVELS FOR 2,3,7,8-TETRACHLORODIBENZO-p-DIOXIN (2,3,7,8-TCDD)

Government	Experimental Basis	NOAEL[2] pg/kg/d	Safety Factor	ADI[3] pg/kg/d
Ontario	Kociba (1978)	1000	100	10
	Murray (1979)	1000	100	10
Netherlands	Kociba (1978)	1000	250	4
Denmark	Kociba (1978)	1000	200	5
Germany	Kociba (1978)	1000	100-1000	1-10

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[1] pg/kg/d: picograms (10^{-12}g) per kilogram body weight per day

[2] NOAEL: No-observed-adverse-effect level in laboratory animals

[3] ADI: Acceptable Daily Intake Value, calculated by dividing the NOAEL by the safety factor

covalently with nucleophilic sites in the cell such as DNA, RNA and protein (Miller and Miller 1966; Miller 1978).

- Many chemical carcinogens display genotoxic activity (McCann et al. 1975). That is, they cause mutations (heritable changes in DNA function) or other effects (such as induction of sister chromatid exchanges, or DNA repair) indicative of interaction with, and alteration of, the normal structure and function of cellular DNA.

- Cancer is heritable at the cellular level, i.e., isolated cancer cells maintain their malignant properties which are passed to the daughter cells when the cancer cell divides.

  In addition, some studies indicate that all of the cancer cells within a cancer appear to be descended from a single initial cell rather than from a mass of cells. This has been demonstrated by the expression of only a single form of the enzyme glucose-6-phosphate dehydrogenase in tumors from women heterozygous for two forms of the enzyme, and who express only one form in each individual cell, but different forms in different cells (Fialkov 1974, 1977). A single-cell origin of chemically-induced tumors has also been documented (Iannaccone et al. 1978). This suggests that cancer represents the expression of a heritable, genetic alteration.

- In the case of ultraviolet (UV) light, both its carcinogenic effects and its mutagenic effects are enhanced in the human disease xeroderma pigmentosum. Individuals with this disease are exquisitely sensitive to sunlight-induced skin cancer (Robbins et al. 1974), and their cells are more sensitive than normal cells to the mutagenic effects of UV light (Maher and McCormick 1976; Maher et al. 1976), and are deficient in the repair of UV light-induced DNA damage. Xeroderma pigmentosum cells in vitro are also more susceptible than normal cells to mutations caused by certain chemical carcinogens. Moreover, UV light-induced carcinogenesis can be prevented by repair (photoreactivation) of specific DNA lesions (pyrimidine dimers) induced by the UV light (Hart et al. 1977).

- For some organ-specific chemical carcinogens, the organ specificity correlates with differences among the organs in repair activity for DNA lesions thought to be precursors of mutagenesis and carcinogenesis (Goth and Rajewsky 1974).

These findings implicate DNA damage as being involved in the process of cancer development, though the exact nature of the change(s) in DNA structure involved is not clear.

G.2.1 INITIATORS AND PROMOTERS

One of the most important concepts in the mechanism of carcinogenicity, initiation and promotion, derives from work on mouse skin carcinogenesis carried out in the 1940s (Rous and Kidd 1941; Berenblum 1941; Mottram 1944; Berenblum and Shubik 1949). These studies revealed that the development of tumors induced by a carcinogen administered at a dose known to be too low by itself to produce a detectable increase in tumor incidence could be enhanced by subsequent repeated treatment with a chemical which was itself without carcinogenic effect when administered alone, even at high, repeated doses.

The initial treatment was termed initiation and the subsequent treatment was termed promotion. Initiation appeared to cause essentially irreversible changes in the skin cells, since enhancement of the carcinogenic effects was seen even if the treatment with the promoter was delayed for several months after initiation (Berenblum and Shubik 1949). The promotional phase was apparently reversible, however, since the effect could be abolished if the interval between repeated treatment was increased. Also, the promoter was without effect if administered prior to the initiator.

Subsequent work has expanded on the concept of initiation and promotion and extended it to sites other than the skin of the mouse, including (with varying degrees of support) liver, lung, colon, bladder, mammary gland, stomach, esophagus, pancreas, and several in vitro systems (Slaga 1983).

The terms initiation and promotion are purely operational; the actual processes involved are not known for certain. However, the most widely accepted hypothesis for the nature of the initiation step is that it involves a heritable alteration in DNA structure, i.e., a mutation. This is consistent with the fact that all known initiators are also capable of causing DNA damage and that the initiation step is relatively irreversible (Farber and Cameron 1980; Slaga 1983). The nature of promotion is even less clear. Hypotheses as to the mechanism of tumor promotion are discussed later.

As noted above for the terms initiation and promotion, so the terms initiator and promoter are operational definitions which refer to particular experimental systems under particular conditions of exposure. Thus, all known skin tumor initiators are also capable of acting as tumor promoters under the appropriate conditions (Slaga 1983). Some tumor promoters can increase the incidence of tumors when administered chronically, though it may be argued that this simply reflects promotion of spontaneous initiating events. Chemicals with both tumor initiating and tumor promoting activity, which can increase tumor incidence when applied alone, are termed "complete carcinogens."

Part of the definition of a tumor promoter is that it can be effective when applied after an initiating treatment, but not before. Other modulators of carcinogen activity are effective when applied chronically at the same time as the carcinogen. Such chemicals are termed cocarcinogens.

G.2.2 GENOTOXIC AND NONGENOTOXIC MODULATORS OF CANCER INCIDENCE

As already discussed, the terms initiation and promotion are operational terms arising out of empirical observations in particular experimental systems involving sequential treatments. Other classifications of chemicals that can influence tumor incidence are possible, however. One such method of classification is that described by Weisburger and Williams (1980). Under this scheme, carcinogens are divided into two major groups: those that interact with and alter DNA, genotoxic carcinogens; and those that do not interact directly with DNA -- nongenotoxic, or epigenetic carcinogens.

Under this scheme, genotoxic carcinogens include chemicals such as alkylating agents (e.g., bis(chloromethyl)ether) which interact directly with DNA; chemicals such as vinyl chloride or benzo(a)pyrene which must be metabolized to an active form before interacting with the DNA; and some inorganic chemicals such as nickel salts which do not interact with DNA, but interfere with the fidelity of DNA replication.

The nongenotoxic or epigenetic group consists of those chemicals that increase tumor incidence without, apparently, directly causing changes in the structure of DNA. This group, as defined by Weisburger and Williams (1980), comprises solid-state carcinogens (such as plastic or metal foils), hormones (such as diethylstilbestrol), immunosuppressors (such as azathioprene), cocarcinogens (such as TPA or catechol), and promoters (such as TPA or phenobarbital).

G.2.3 HYPOTHETICAL MECHANISMS OF CANCER "INDUCTION" BY NONGENOTOXIC CARCINOGENS

Several different hypotheses have been proposed to explain the action of non-genotoxic carcinogens. Several suggest that non-genotoxic carcinogens act by enhancing the activity of naturally occurring genotoxic carcinogens or spontaneous genetic damage. This enhancement may occur by increasing the amount of DNA damage caused by a genotoxic carcinogen, for example, by increasing the proportion of a procarcinogen that is metabolized to an active form; by reducing the rate of metabolic inactivation or clearance of an active carcinogen; by interfering with the repair of the DNA damage caused by the genotoxic carcinogen; or in some other manner increasing the expression of induced or spontaneous genetic damage.

Characteristics of Tumor Promoters

Hypotheses as to the nature of promotion derive from theoretical concepts, observations of developing tumors, and observations of the biological effects of chemicals with strong tumor-promoting activity, such as 12-O-tetradecanoylphorbol-13-acetate (TPA). Farber and Cameron (1980) note that:

> "the essence of promotion is the selective stimulation of initiated cells to grow relative to surrounding cells; i.e., it is the creation of a differential growth environment, leading to amplification of the initiated cell population."

Table G-2 lists the effects of TPA and other tumor promoters in mouse skin. Of the effects listed, three stand out as particularly important. First, all known skin tumor promoters induce epidermal hyperplasia (Slaga 1983), though not all chemicals that induce epidermal hyperplasia are skin tumor promoters (Slaga et al. 1975). Second, there is a strong correlation between the ability to induce the activity of the enzyme ornithine decarboxylase (ODC) in mouse skin and tumor

TABLE G-2

MORPHOLOGICAL AND BIOCHEMICAL RESPONSES OF MOUSE SKIN TO PHORBOL ESTER AND OTHER TUMOR PROMOTERS (SLAGA 1983)

Induction of inflammation and hyperplasia

Increase in DNA, RNA and protein synthesis

Initial increase in keratinization followed by a decrease

Increase in phospholipid synthesis

Increase in prostaglandin synthesis

Increase in histone synthesis and phosphorylation

Increase in ornithine decarboxylase activity followed by increase in polyamines

Increase in histidine and DOPA decarboxylase activity

Decrease in the isoproterenol stimulation of cAMP

Decrease in the number of dexamethasone receptors

Decrease in SOD and catalase

Induction of embryonic state in adult skin

1. Induction of dark cells (primitive stem cells)
2. Induction of embryonic proteins in adult skin
3. Induction of morphological changes in adult skin resembling papillomas, carcinomas and embryonic skin
4. Decrease in histidase activity
5. Increase in protease activity
6. Decrease in response of G_1 chalone in adult skin
7. Increase in cAMP independent protein kinase in adult skin resembling tumors and embryonic skin

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promoting activity (O'Brien et al. 1975), though the weak promoter mezerein is similar to the strong promoter TPA in its ability to induce ODC activity (Mufson et al. 1979). Third, tumor promoters appear to induce what resembles an embryonic state in adult skin. This includes the appearance of "dark basal cells" in the epidermis. This effect differentiates tumor promoters like TPA from chemicals such as ethylphenylpropiolate which causes epidermal hyperplasia, but is not a tumor promoter in mouse skin (Raick and Burdzy 1973), and from chemicals like mezerein which is a potent inducer of ODC activity but only a weak promoter (Mufson et al. 1979).

Interpretation of the mechanism of action of tumor promoters is complicated by the suggestion by some (Slaga 1983; Furstenberger et al. 1981) that tumor promotion is at least a two-stage process. This suggestion is based on the finding that some chemicals, such as mezerein, which, by themselves are weak promoters, produce a large promoting effect if initiation is followed by a brief treatment with TPA prior to prolonged treatment with mezerein. This interpretation has been challenged by Hennings and Yuspa (1985).

The initiation-promotion concept has been extended from mouse skin to other sites, notably the liver (Herren and Pereira 1983) and the urinary bladder (Cohen 1983). Even less is understood about the mechanism of promotion in these organ systems than in skin, though, as with mouse skin, enhanced cell proliferation appears to play a role (Cohen 1983; Goldfarb et al. 1983).

Induction of Cell Proliferation

Just as the induction of cell proliferation (hyperplasia) seems a common activity of chemicals active as mouse skin tumor promoters, so it has been proposed that the induction of cell proliferation may be involved with other nongenotoxic carcinogens. Reitz et al. (1980) have suggested that tissue damage leading to regenerative hyperplasia is involved in the mechanism of action of some nongenotoxic carcinogens such as carbon tetrachloride.

During normal DNA replication, it is likely that errors are made at a very low, but not zero, rate. Increasing the amount of DNA replication, as would occur during hyperplasia, would increase the total number of replication errors. The effect of such an error might be analogous to that of a chemically-induced DNA lesion produced by a genotoxic carcinogen. Alternatively (or additionally) the increased rate of DNA replication would give less time for DNA repair systems to repair damage caused by the "natural" low level of genotoxic chemicals (aflatoxin, nitrosamine, polycyclic aromatic hydrocarbons) or physical agents (ultraviolet light, X-rays, γ-rays) in the environment. Such a model might also explain the mode of action of carcinogenic hormones such as estrogens (Hertz 1977, Shellenberger 1980, Huseby et al. 1980, Drosdowsky et al., 1980) which stimulate DNA synthesis and cell division in their target organs.

Some support for this hypothesis comes from the finding of Berman et al. (1978), that increasing the rate of DNA replication in normal cells in tissue culture increases their susceptibility to mutagens. Other data, however, argue against this idea. If an elevated rate of cell replication were itself carcinogenic, one might expect a high incidence of cancer during embryonic and fetal development and early childhood when cell replication is very rapid. In fact, the cancer incidence is low at this stage of life; though it may be argued that an elevated cancer incidence would ensue only if the elevated replication rate were continued for a lifetime.

Recently, Butterworth (1984) has proposed that the induction of DNA replication may explain the carcinogenic activity of di(2-ethylhexyl)phthalate (DEHP) and perhaps other agents that cause peroxisome proliferation since DEHP induces cell replication in the liver, the site of tumor formation.

The exact relationship between increased cell proliferation and cancer is far from clear. However, some results strongly suggest that some carcinogens exert their effects by increasing cell proliferation (at least in the mouse liver). Mirsalis et al. (1985) examined seven liver carcinogens for the capacity to cause unscheduled DNA synthesis (UDS, a measure of genetic damage) and to cause cellular proliferation. Four of the carcinogens caused no increase in UDS but caused significant increases (>100-fold) in cell proliferation.

Mirsalis (1986) stated that his data convince him that increased cellular proliferation is sufficient to cause liver tumors in mice. He is not certain about the relationship between cell proliferation and carcinogenesis in rat liver. In that organ, some agents that cause proliferation cause cancer; others do not. Other scientists (among them, Farber 1984; Harris and Sun 1984) have also explored the relationship between cellular toxicity, compensatory proliferation, and cancer.

Peroxisome Proliferation

A number of structurally unrelated chemicals which share the biological properties of causing proliferation of the cell organelles, peroxisomes, and causing liver enlargement and hypolipidemia, also share the property of causing liver cancer in rodents (Reddy and Lalwani 1983). None of these chemicals is genotoxic, and several hypotheses have been proposed to explain their carcinogenicity. Most prominent among this is the suggestion by Reddy and coworkers (Reddy et al. 1980; Warren et al. 1982) that cancer induction is a result of protracted proliferation of peroxisomes and induction of peroxisomal enzymes, leading to elevated levels of hydrogen peroxide (a byproduct of several of the peroxisomal enzymes including one involved in oxidation of fatty acids). It is hypothesized that the elevated activity of fatty-acid oxidizing enzymes causes the hypolipidemic effect, and that the byproduct, hydrogen peroxide, which is produced in excessive amounts (Lalwani et al. 1981; Reddy et al. 1979; Tomaszewski et al. 1985), is responsible for the carcinogenic effect, either directly, or via lipid oxidation (Ames 1983; Cerutti 1985).

At least partial support for this hypothesis comes from the demonstration of increased levels of hydrogen peroxide in liver of rodents treated with peroxisome proliferators (Lalwani et al 1981; Reddy et al 1979; Fahl et al. 1984; Tomaszewski et al. 1985); by increased levels of lipofuscin and other indicators of lipid peroxidation in tissues of animals treated with peroxisome proliferators (Reddy et al. 1982; Lake et al. 1986); and by the inhibitory effects of the antioxidant ethoxyquin on liver tumor incidence in rats fed the peroxisome proliferator ciprofibrate (Rao et al. 1984).

This mechanism is far from proven, however, and an alternative explanation that peroxisome proliferators act by increasing cell replication has been proposed by Butterworth (1984) as noted above.

Immunosuppression

The idea that the immune system might be involved in protecting against cancer development was initially proposed by Paul Ehrlich in 1909 (Herberman 1985). The immune surveillance hypothesis subsequently developed by Thomas (1959) and Burnet (1957; 1970) elaborated on this suggestion and has been the subject of considerable research and controversy.

The basic thesis of the immune surveillance hypothesis is that the immune system is continually monitoring the body for cells that are foreign or abnormal (like tumor cells), and such cells are killed by the immune system, particularly the T-cell mediated immune system which normally acts to destroy foreign cells. If the immune system is suppressed, tumor cells arising spontaneously may escape this surveillance and multiply to form a tumor.

Support for the theory comes from the finding that, in some cases, naturally occurring, or induced, immunosuppression is associated with an elevated incidence of some types of tumors, particularly those induced by tumor viruses in animals (Herberman 1985). In mice, the susceptibility to urethane-induced tumors among different strains is correlated with the degree of inhibition of the immune system, particularly the natural killer (NK) cells induced by urethane in those different strains (Gorelik and Herberman 1981). Also, human patients treated with immunosuppressive agents, particularly prednisone and azathioprene, show an increased incidence of certain types of cancer (Penn 1985); and the high incidence of Kaposi's sarcoma among patients with acquired immune deficiency syndrome (AIDS) provides additional support for the concept.

Other data, however, are not consistent with a simple model of immune surveillance: most chemically induced tumors (unlike virally induced tumors) are not strongly antigenic and tend not to stimulate an immune response. The tumors seen in humans associated with immunodepression tend to be leukemias and lymphomas, rather than a broad range of common tumors (Herberman 1985), and there is not a consistent association between immunodepression and cancer. For example, athymic nude mice, which lack a thymus gland and are severely deficient in cell-mediated immunity, show a pattern and incidence of tumors similar to normal mice (Stutman 1975). In the case of nude mice, however there is evidence that they have enhanced levels of natural killers (NK) cells which may compensate for the lack of T-cells in immune surveillance (Benjamini et al. 1982).

G.3 EVIDENCE THAT 2,3,7,8-TCDD IS PROBABLY NOT A GENOTOXIC TUMOR INITIATOR

Evidence that 2,3,7,8-TCDD is probably not a genotoxic tumor initiator comes from two sources: tests of the activity of 2,3,7,8-TCDD in assays for mutagenicity and other indicators of genotoxicity (e.g. DNA repair assays); and tests of the activity of 2,3,7,8-TCDD in in vivo and in vitro tumor initiation/promotion assays.

G.3.1 ASSAYS IN BACTERIA AND YEAST

Of seven studies using various strains of *Salmonella typhimurium* to detect reverse mutations from histidine requirement to histidine independence, only two early studies, those by Hussain et al. (1972) and Seiler (1973), showed any sign of positive results. In both studies, a positive response was noted in strain TA1532, a strain which detects frameshift mutations. Seiler (1973) used a spot-test, a crude screening test in which a crystal or drop of a test material is placed in a dish containing bacteria, and an increase in the number of colonies of bacteria growing up around the test material is considered an indication of mutagenicity. The results observed by Seiler (1973) are most likely an artifact since the spot-test relies on diffusion of the test material through the agar of the test plate to expose the bacteria. The extremely low water solubility of 2,3,7,8-TCDD probably would have prevented diffusion and hence prevented exposure of the bacteria. Further, when McCann (unpublished, cited by Wassom 1977) repeated the spot-test with 2,3,7,8-TCDD and the same strain of Salmonella, no indication of a positive response was seen.

Hussain et al. (1972) incubated the bacteria in liquid suspension prior to testing them for increased reversion frequency. A significant increase in reversion frequency (not dose-related) was reported at 2,3,7,8-TCDD concentrations of 2-3 µg/ml which caused more than 50% cell death. Importantly, the apparent positive effect occurred at 2,3,7,8-TCDD concentrations above its level of water solubility and at a level associated with poor bacterial survival. Since other studies using strain TA1532 (McCann, cited by Wassom 1977; Gilbert et al. 1981) have not confirmed the findings of Sieler (1973) and Hussain et al. (1972), and since there are technical problems which cast doubt on the validity of their conclusions, the studies of Seiler (1973) and Hussain et al (1972) cannot be considered as providing clear evidence for the mutagenicity of 2,3,7,8-TCDD. Furthermore, one of the co-authors (G. Lofroth) of the Hussain et al. (1972) paper has since stated that ". . . it is apparent that TCDD shows no or very little mutagenic activity in in vitro test systems such as the Ames Salmonella test." (Toftgard et al. 1983).

The seven studies of the mutagenicity of 2,3,7,8-TCDD in Salmonella have used a total of 13 different strains of the bacteria, 6 sensitive to base-pair substitution mutations and seven sensitive to frameshift mutations (Hussain et al. 1972; Seiler 1973; Nebert et al. 1976; McCann, cited Wassom et al. 1977; Geiger and Neal 1981; Gilbert et al. 1981; Mortelmans et al. 1984). None of the studies other than those by Hussain et al. (1972) and Seiler (1973) have demonstrated any indication that 2,3,7,8-TCDD is mutagenic, either by base-pair substitution, or by frameshift, either in the presence, or in the absence, of rodent liver microsomal metabolic activation systems.

Hussain et al. (1972) also tested 2,3,7,8-TCDD for its ability to induce reversion to streptomycin independence in E. coli Sd-4 bacteria, and to induce prophage in E. coli K-39. Again, "positive" results were reported only at concentrations above the solubility limit, which were extremely toxic to the bacteria. Because of these technical problems, these cannot be considered valid tests of the mutagenicity of 2,3,7,8-TCDD.

Bronzetti et al. (1983) measured mitotic gene conversion and reverse gene mutation in cells of the yeast Saccharomyces cerevisiae strain D-7. The cells were exposed to 2,3,7,8-TCDD at 0.5 to 10 µg/ml in suspension in the presence and absence of a rodent liver metabolic activation system. Small, dose-related increases (three-fold or less) in both reverse mutation and gene conversion were seen in the presence, but not in the absence, of the metabolic activation system. The apparent increases in mutation and gene conversion frequency were accompanied by reduction in cell survival, and there was no absolute increase in the number of mutants or convertants. Therefore, one possible explanation for these results is that the observed effect is due to a preferential survival of preexisting spontaneous mutants and convertants rather than induction by 2,3,7,8-TCDD.

Bronzetti et al (1983) also reported that injection of a single dose of 2,3,7,8-TCDD at 25 µg/kg in male Swiss albino mice into which yeast cells had been injected i.v. resulted in a two- to three-fold increase in gene conversion frequency and in mutation frequency in yeast cells harvested from the liver and kidneys 20 days later. The very long delay between administration of the chemical and cells and harvesting of the cells, and the small magnitude of the increase in mutation and gene conversion frequency raise questions about the relevance of these observations to the toxicity of 2,3,7,8-TCDD.

G.3.2 ASSAYS IN MAMMALIAN CELLS IN VITRO

Assays for the Induction of Drug-Resistance

Rogers et al. (1982) have tested 2,3,7,8-TCDD for mutagenicity in L5178Y mouse lymphoma cells using resistance to methotrexate, excess thymidine, thioguanine, ouabain and cytosine arabinoside as markers. A dose-related increase in the frequency of colonies resistant to methotrexate, thymidine or thioguanine was observed, but the frequency of colonies resistant to ouabain or cytosine arabinoside was not changed. The increase occurred with 2,3,7,8-TCDD concentrations of 0.1 to 0.5 μg/ml at survival levels of 87% to 75%. Whether these increases represent true increases in mutation frequency is unclear, however. For example, it is possible that the 2,3,7,8-TCDD treatment enhances metabolic inactivation of the selective agents, thus resulting in increased survival of colonies which do not represent true mutants. Furthermore, the tested concentrations were well above the solubility of 2,3,7,8-TCDD, which is 2×10^{-5} μg/ml (Marple et al. 1986).

Unscheduled DNA Synthesis

Two studies have examined the ability of 2,3,7,8-TCDD to induce DNA repair as measured by unscheduled DNA synthesis (UDS), a sensitive measure of the ability of a chemical to cause DNA damage. Loprieno et al. (1982) observed no increase in UDS in human cells treated in culture with 2,3,7,8-TCDD. More importantly, Althaus et al. (1982) observed no increase in UDS in cultured primary rat hepatocytes which are capable of metabolizing 2,3,7,8-TCDD and which represent target cells for 2,3,7,8-TCDD's carcinogenicity.

Cell Transformation

Two studies have also examined the ability of 2,3,7,8-TCDD to induce cell transformation. Hay et al. (1983) used the cell line BHK21 C13 derived from hamster kidney cells and observed a dose-related increase in the proportion of surviving cells which were able to grow in semi-solid agar. This work was briefly described in an abstract of a meeting of the American Chemical Society. No paper describing the work has yet appeared in a peer-reviewed journal, and Robert Neal (personal communication) has been told that there are no plans to publish such a paper. Therefore there is no way to examine the specifics of the experiment.

Abernethy et al. (1985) used the mouse C3H/10T½ cell line and found no increase in the number of transformed colonies when cells were treated with 2,3,7,8-TCDD alone, though they did report an enhancement of transformation induced by the mutagen N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) when

2,3,7,8-TCDD treatment followed MNNG treatment. Abernethy et al. (1985) suggested that this was a demonstration of the promotional ability of 2,3,7,8-TCDD. It is possible that, since the BHK cells used by Hay et al. (1983) are partly transformed, the positive results reported by them represent a promoting rather than an initiating effect of 2,3,7,8-TCDD.

In Vitro Sister Chromatid Exchange Assay

Toth et al. (1984) tested 2,3,7,8-TCDD at 10^{-4} μg/ml for its ability to induce sister chromatid exchanges in cultured Chinese hamster cells and observed negative results. Although this concentration is low compared to those tested by others, it is above the level of solubility of 2,3,7,8-TCDD in water of 2×10^{-5} μg/ml (Marple et al. 1986).

G.3.3. ASSAYS IN INTACT ANIMALS

2,3,7,8-TCDD has been tested for the capacity to cause mutations in fruit flies and in rats, and for binding to DNA in rat liver DNA. No positive results have been observed.

Drosophila

Zimmering et al. (1985) tested 2,3,7,8-TCDD for activity in the sex-linked recessive lethal mutation assay in Drosophila.

2,3,7,8-TCDD was administered by injection at 50, 250, or 500 ppm in peanut oil. These treatments induced up to 27% lethality in the treated flies but caused no increase in recessive lethal mutations.

Rodent Dominant Lethal Assay

Khera and Ruddick (1973) administered 2,3,7,8-TCDD orally to male rats at 4, 8, or 12 mg/kg/day for 7 days. Although signs of toxicity (and some deaths) were seen in treated animals, no increase in dominant lethal mutations was observed.

DNA Binding Assay

Poland and Glover (1979) measured the amount of binding of 2,3,7,8-TCDD (or 2,3,7,8-TCDD metabolities) to DNA in the liver of rats given a single intraperitoneal injection of ^{3}H-2,3,7,8-TCDD (7.5 μg/kg) and killed 12 hours, 48 hours, or 7 days later. The amount of radioactivity that remained associated with the DNA after isolation and purification was very low, and probably of no biological significance. On a molar basis, 2,3,7,8-TCDD was bound between 10,000 and 100,000-times less than genotoxic carcinogens (such as 2-acetylaminofluorene or benzo(a)pyrene) after similar treatment (Poland and Glover 1979; Farber 1968).

Cytogenetic Studies

Several studies have examined the ability of 2,3,7,8-TCDD to induce chromosome aberrations in bone marrow cells in rats and mice (Green and Moreland 1975; Green et al. 1977; Loprieno et al.1982; Meyne et al. 1985). Of these four studies, two, those by Green et al. (1977) and Loprieno et al. (1982) have reported increases in aberration frequency.

Green et al. (1977) examined the frequency of cells with chromosome aberrations in the bone marrow of Osborne Mendel rats that had received 2,3,7,8-TCDD at 0.25, 0.5, 1.0, 2.0, or 4.0 µg/kg by gavage in acetone/corn oil (1:9) twice per week for 13 weeks. The frequency of cells with "aberrations" (not specified) at the highest dose level in females and at the two highest dose levels in males was significantly greater than at the lowest dose level (no control group was included). However, the facts that no control group was included, that the apparent increase in aberrations occurred only at grossly toxic dose levels, and that the frequency of aberrant cells in the low dose males was unusually low (0.47%) raises questions about the biological significance of these findings.

Loprieno et al. (1982) reported an increase in the frequency of chromatid gaps and aberrations in bone marrow cells of mice given a single i.p. dose of 2,3,7,8-TCDD at 10 µg/kg, but not in female rats given 2,3,7,8-TCDD at 0.01, 0.1, or 1.0 µg/kg once per week for 45 weeks.

In contrast to these apparently weak positive results, Meyne et al. (1985) found no increase in chromosome damage in bone marrow cells of responsive (C57Bl/6J) or nonresponsive (DBA/2J) mice given doses of 2,3,7,8-TCDD (up to 150 µg/kg) that were grossly toxic and caused liver damage in the treated animals. Nor did Green and Moreland (1975) observe increases in chromosome damage in rats given 2,3,7,8-TCDD by gavage at 10 µg/kg/day for 5 days or 20 µg/kg as a single dose, or by i.p. injection as a single dose of 5, 10, or 15 µg/kg.

Taken as a whole, currently available data do not provide clearly positive evidence that 2,3,7,8-TCDD causes structural chromosome damage in rats or mice.

G.3.4 CYTOGENETIC STUDIES IN HUMANS

Several studies have examined the frequency of chromosome aberrations in humans exposed to 2,3,7,8-TCDD. This includes two studies of chemical workers (Czeizel and Kiraly 1976; Hay 1977), and a large number of studies of individuals exposed as a result of the accident in Seveso, Italy during which a reaction vessel in a chemical plant exploded and liberated a large quantity of chemicals, including 2,3,7,8-TCDD, over the surrounding countryside (Wassom et al. 1977; Hay 1977, 1982; Reggiani 1979, 1980; Pocchiari et al. 1979; Tenchini et al. 1979, 1983; Homberger et al. 1979; Mottura et al. 1981; DiLernia et al. 1982; Tenchini 1983).

The two studies of chemical workers gave conflicting results, with an increase in aberrations reported by Czeizel and Kiraly (1976) and no increase reported by Hay (1977). The significance of the increase reported by Czeizel and Kiraly (1976) is questionable because of the exposure of the individuals to chemicals other than TCDD. This is particularly true in light of the negative data reported by Hay (1977) in which 800 individuals with chloracne due to TCDD exposure were studied.

The studies of individuals in Seveso reveal a generally negative picture. The only indications of positive results come from a preliminary study by Tenchini et al. (1979) which was subsequently shown to be artifactual and not due to TCDD exposure (Tenchini et al. 1983), and a study by DiLernia et al. (1982) which examined not structural chromosomal aberrations but changes in satellite association pattern. This endpoint is not, however, generally recognized as an indicator of genotoxicity. All other studies cited above showed no significant increase in chromosome aberration frequency among cells of individuals exposed to TCDD at Seveso.

G.3.5 CONCLUSIONS CONCERNING GENOTOXICITY OF 2,3,7,8-TCDD

When viewed as a whole, the genotoxicity data on 2,3,7,8-TCDD do not provide convincing evidence that 2,3,7,8-TCDD is capable of causing genetic damage. The positive studies sited above all have limitations. It is still possible, however, that 2,3,7,8-TCDD, under some conditions, may have a genotoxic effect. For example, S. Safe (personal communication) in collaboration with Kurt Randerath has preliminary evidence that 2,3,7,8-TCDD causes increased DNA adduct formation with "endogeneous initiators" being the adducts. Those events are not direct interactions between an exogeneous agent (in this case 2,3,7,8-TCDD) and DNA, but are indirect events. Should the results be verified, they are compatible with a different pathway to DNA adduct formation and not evidence for 2,3,7,8-TCDD, directly or by itself, being genotoxic; in fact, it is consistent with a promotional mechanism. Estrogens play a similar role in promoting adduct formation from endogeneous initiators.

G.3.6 TESTS FOR INITIATION

In only one study reported in the literature has 2,3,7,8-TCDD been tested as a initiator in an initiation/promotion assay in vivo. DiGiovanni et al. (1977) applied a single dose of 2,3,7,8-TCDD (2 μg per mouse) to the shaved back of female CD-1 mice and after a one-week interval applied TPA (5 μg/mouse) in acetone twice weekly for 32 weeks. At the end of this time, 14% of the mice had papillomas. Because no control groups receiving only 2,3,7,8-TCDD or only TPA were employed, these results cannot be clearly interpreted.

2,3,7,8-TCDD has also been tested in an in vitro study examining cell transformation in C3H mouse 10T½ cells (Abernethy et al. 1985). Cells were treated with a range of 2,3,7,8-TCDD concentrations (0 to 5,000 nM) or with 3.4 μM MNNG for 24 hours. The medium was then replaced with medium containing TPA at 0, or 0.25 μg/ml. 2,3,7,8-TCDD treatment did not cause an increased incidence of cell transformation, either with or without TPA. In contrast, the TPA treatment substantially enhanced (by about 16-fold) the frequency of cell transformation in the culture that had received the direct-acting mutagen MNNG.

The data described above do not provide substantial and definitive evidence that 2,3,7,8-TCDD is capable of acting as a genotoxic initiator of carcinogenesis. This suggests that whatever the mechanism by which 2,3,7,8-TCDD causes cancer in rodents, it is not likely to be a genotoxic initiator, at least as we currently know them to behave.

G.3.7 EVIDENCE FOR 2,3,7,8-TCDD BEING A TUMOR PROMOTER

Poland et al. (1982) have shown that 2,3,7,8-TCDD acts as a skin tumor promoter in HRS/J mice homozygous for the recessive hairless gene (hr/hr); while it is inactive in heterozygous (hr/+) mice. In hairless mice, a single application of 5 μmoles of MNNG or 0.2 μ moles of dimethylbenz(a)anthracene

(DMBA) followed by twice weekly application of 2,3,7,8-TCDD (3.75 - 50 ng/mouse) resulted in an increased incidence of skin tumors compared to animals treated only with that initiator (MNNG or DMBA) or only with 2,3,7,8-TCDD. For example, mice receiving only the MNNG treatment had a skin tumor incidence 20 weeks later of 1/19 and mice receiving only twice-weekly treatment with 2,3,7,8-TCDD at 30 ng/mouse had a skin tumor incidence of 0/19. Mice receiving both chemicals, however, developed skin tumors. At 30 ng 2,3,7,8-TCDD, 15 out of 19 mice developed tumors; at 15 ng, 10 out of 10 developed tumors. Even at a 2,3,7,8-TCDD dose level of 3.75 ng/mouse the tumor incidence was 11/20. In this strain of mice, 2,3,7,8-TCDD was 100 times as effective as a promoter of skin tumors as the classical skin tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA).

More importantly, 2,3,7,8-TCDD has been shown to act as a promoter of liver tumors in rats of normal genetic constitution. Pitot et al. (1980) subjected female Sprague-Dawley rats to 70% partial hepatectomy, and administered a single initiating dose of diethylnitrosamine (DEN) at 10 mg/kg by gastric intubation 24 hours later. Control groups received water instead of DEN. Starting one week later, groups of initiated and control rats received biweekly subcutaneous injections of 2,3,7,8-TCDD in corn oil at 0.14 or 1.4 μg/kg, or were administered sodium phenobarbital at 0.05% in the diets for 28 weeks. The animals were then killed and sections of their liver examined for tumors and foci of altered enzymic activity.

Rats subjected to partial hepatectomy plus DEN displayed many small enzyme-altered foci, but no tumors. Rats subjected to partial hepatectomy plus 2,3,7,8-TCDD or phenobarbital alone showed only small numbers of foci and no tumors. No foci were seen in rats following only partial hepatectomy and no chemical. Both 2,3,7,8-TCDD and phenobarbital, when administered after DEN, enhanced the number and size of the enzyme-altered foci and led to the development of tumors. The results of the study are summarized in Table G-3.

Although these studies demonstrate that 2,3,7,8-TCDD can act as a "promoter," the implications of this finding for the understanding the carcinogenic mechanism of action of 2,3,7,8-TCDD is unclear. On the basis of their work, Pitot et al. (1980) suggested that when 2,3,7,8-TCDD is administered alone in a lifetime bioassay, it may be "promoting cells initiated by ambient environmental conditions such as diet, background or other factors... and giving its effects the semblance of a complete carcinogen."

It is not at all clear that all promoters have the same mechanism of action. This is highlighted by the comments of

TABLE G-3

PROMOTING EFFECTS OF 2,3,7,8-TETRACHLORODIBENZO-p-DIOXIN (2,3,7,8-TCDD) ON HEPATOCARCINOGENESIS BY A SINGLE DOSE OF DIETHYLNITROSAMINE (DEN) AND PARTIAL HEPATECTOMY (PH).

(PITOT et al. 1980).

Treatment	n[a]	Number of enzyme-altered foci per cm^3 of liver	Mean volume of enzyme-altered foci (mm^3)	Volume occupied by foci (%)	Number of rats with carcinoma
PH only	?	0	0	0	0
PH + DEN	4	309 ± 98	0.02	0.7	0
PH + TCDD (low dose)	4	34 ± 17	0.05	0.2	0
PH + TCDD (high dose)	5	25 ± 7	0.04	0.1	0
PH + Phenobarbital	4	56 ± 13	0.01	0.1	0
PH + DEN + TCDD (low dose)	5	1068 ± 166	0.08	9.0	0[b]
PH + DEN + TCDD (high dose)	7	871 ± 66	0.49	43.0	5[c]
PH + DEN + Phenobarbital	10	533 ± 103	0.15	6.0	8

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[a] n is the number of animals per group; the number in the PH-only group was not reported.

[b] Three rats had neoplastic nodules.

[c] One other rat had a neoplastic nodule.

Poland et al. (1982) that the biological effects of 2,3,7,8-TCDD on the skin of the hairless mouse in which it acts as a promoter are quite different from the effects of the classic promoter TPA. The latter produces acute inflammation and hyperplasia of the epidermis. 2,3,7,8-TCDD, however, produces no inflammation and is systemically more toxic than TPA. 2,3,7,8-TCDD also causes epidermal hyperplasia and hyperkeratosis, squamous metaplasia of the sebaceous glands, and accumulation of keratinaceous material in dermal cysts in hairless mice, but has no gross or histological effects on the skin of normal haired mice in which 2,3,7,8-TCDD has no promoting activity. This fact, plus limited investigation of the activity of other structurally-related halogenated hydrocarbons in the hairless mouse skin tumor promotion assay, has led Poland et al. (1982) to suggest that the mechanism of tumor promotion by 2,3,7,8-TCDD and related chemicals is different from that of TPA, and that the mechanism of tumor promotion of 2,3,7,8-TCDD and related chemicals is mediated through the Ah receptor. In contrast, the activity of TPA is apparently mediated through the phorbol diester receptor (a membrane-bound receptor different from the Ah receptor) which is apparently identical to the diacylglycerol receptor of protein kinase C, an enzyme involved in regulation of cell function and differentiation (Niedel et al. 1983; Kikkawa et al. 1983; Takai et al. 1984).

Poland et al. (1982) examined skin-tumor promoting activity of other chlorinated dioxins and furans in hairless mice. The chemicals that bind most strongly to the Ah receptor were the most efficient tumor promoters. These observations reinforce conclusions about the general role of the Ah receptor in the toxicity of 2,3,7,8-TCDD and about its role in promotion.

One effect that TPA and 2,3,7,8-TCDD share in hairless mice is that of causing epidermal hyperplasia. It is possible that this hyperplastic effect is responsible for the promoting effects. As noted above, hyperplasia is an effect common to many tumor promoters (Slaga 1983). Both 2,3,7,8-TCDD and TPA stimulate terminal differentiation of epidermal basal cells in culture causing increased keratin synthesis, formation of cornified envelopes, and increased activity of the enzyme epidermal transglutaminase (Yuspa et al. 1982; Reiners and Slaga 1983; Puhvel et al. 1985; Osborne and Greenlee 1985). Whether this effect is also related to the mechanism of tumor promotion is unclear, however.

G.3.8 INTERACTIONS OF 2,3,7,8-TCDD WITH OTHER CARCINOGENS

In addition to studies in which it has been tested in initiation/promotion protocols, the influence of 2,3,7,8-TCDD on the carcinogenic response to other carcinogens has been examined in several other studies.

Berry et al. (1979) observed that treatment of CD-1 mice with 2,3,7,8-TCDD before treatment with DMBA inhibited the development of skin tumors induced by DMBA and promoted by TPA. The effect of 2,3,7,8-TCDD was greatest when applied 3 to 5 days before the DMBA; under those conditions, inhibition of the tumor response was 94 to 96% complete. 2,3,7,8-TCDD was ineffective, however, when administered only 5 minutes before initiation with DMBA (Berry et al. 1979), or when administered concurrently with DMBA (DiGiovanni et al. 1977). The time course of the inhibitory effect on tumor development paralleled the time course for induction of Aryl Hydrocarbon Hydrogenase (AHH) in the skin. The inhibition was also associated with a reduction in covalent binding of DMBA metabolites to DNA and RNA, but there was no observed effect on binding to protein.

A similar, though not so extreme, inhibitory effect of 2,3,7,8-TCDD was also seen with skin tumor initiation by benzo(a)pyrene (BaP) (Berry et al. 1979). Similar effects of 2,3,7,8-TCDD on skin tumor induction by DMBA and BaP were also reported by Cohen et al. (1979), who noted that pretreatment of Sencar mice with 2,3,7,8-TCDD markedly reduced the formation of the presumptive ultimate carcinogenic metabolite of BaP, 7,8-diol-9,10-epoxy BaP, and its covalent binding to DNA. DiGiovanni et al. (1980) observed a similar pattern of 2,3,7,8-TCDD inhibition of BaP and 3-methylcholanthrene (MCA) induced skin cancer in CD-1 mice. That is, concurrent treatment with 2,3,7,8-TCDD was essentially without effect, while 2,3,7,8-TCDD treatment three days before BaP or MCA substantially inhibited (by 86% and 57%, respectively) the subsequent development of skin tumors.

The similarity of the timing of maximum inhibition of tumor induction to the timing of maximum induction of microsomal enzymes suggests that the effect of 2,3,7,8-TCDD is due to its liver enzyme inducing ability. A different pattern of response, however, has been reported with the interaction of 2,3,7,8-TCDD and MCA by Kouri et al. (1978) in two other strains of mice.

Kouri et al. (1978) examined the effect of prior or simultaneous administration of 2,3,7,8-TCDD (100 µg/kg and 1 µg/kg) on the incidence of injection-site fibrosarcomas (it is unclear if tumor types were seen) induced by MCA in C57Bl/6 Cum and DBA/2 Cum mice which are responsive and nonresponsive, respectively, to the induction of AHH by MCA. (MCA is a complete carcinogen and unlike 2,3,7,8-TCDD, it is genotoxic) In the responsive C57Bl/6 mice, the tumor incidence was high (70-100%) in all groups receiving MCA (with or without 2,3,7,8-TCDD) and no clear influence of 2,3,7,8-TCDD on MCA-induced tumors could be determined. In the non-responsive DBA/2 mice, however, MCA alone produced very few tumors, and concurrent treatment with 2,3,7,8-TCDD produced a dose-related enhancement of tumor incidence. Treatment with 2,3,7,8-TCDD two days before MCA treatment was without effect. 2,3,7,8-TCDD alone did not induce tumors in either strains of mice.

It is unclear whether this different pattern of response is due to the differences in mouse strain, or to the different routes of administration and tumor types (skin tumors following skin painting versus injection-site tumors following subcutaneous injection).

G.4 POTENTIAL MECHANISMS OF TUMOR PROMOTION

As noted above, the process of cancer development appears to be a multistep phenomenon. By following sequentially the development of tumors, either spontaneous or carcinogen-induced, it is possible to infer a sequence of stages of tumor development. Unfortunately, because it is not possible to follow sequentially the fate of all of the cells in an area of tissue in which a tumor ultimately develops, it is not possible to say whether each "stage" which can be differentiated by its appearance reflects a necessary stage of tumor development. Of the stages that are necessary, however, it would be anticipated that a chemical that affected the rate of progress of a cell from any one stage to a subsequent stage, or that increased the number of cells available for progression between stages, would increase the overall rate and incidence of cancer development (unless another stage, unaffected by the chemical was rate limiting in the process of cancer development).

As a result, although much emphasis has been placed on the initiation stage and its effect on cancer incidence, the facts that cancer develops spontaneously in control animals and humans not known to be exposed to carcinogens (i.e., spontaneous initiation presumably does occur), and that the incidence of cancer among an initiated population can be greatly enhanced by "promoters" or other modifiers of cancer development, indicate that other stages of cancer development are important in determining the overall incidence of cancer.

In the absence of clear evidence that 2,3,7,8-TCDD is an initiator, and since there is some evidence that it can act as a tumor promoter (see above), it is of interest to consider possible mechanisms by which these effects may be expressed.

G.4.1 INCREASED PROLIFERATION OF SPONTANEOUSLY INITIATED CELLS

As already noted, the induction of cell proliferation has been proposed in the past as a mechanism of cancer causation. Nongenotoxic chemicals could cause an increase in DNA synthesis with a concomitant increase in the number of errors in DNA replication, or they could limit the time available for repair of spontaneous DNA damage, and/or stimulate the division of initiated cells or cause any combination of these effects simultaneously.

There is some circumstantial evidence that hyperplasia may be involved in the development of cancer following exposure to 2,3,7,8-TCDD. In the rat, two of the target organs for carcinogenicity are liver and lung (Kociba et al. 1978). Rat liver is known to contain many spontaneous preneoplastic cells that can be stimulated by promoting agents to produce tumors (Ogawa et al. 1981; Ward 1983; Schulte-Hermann et al. 1983), and both liver and lung respond to exposure to 2,3,7,8-TCDD with hyperplasia (Poland and Knutson 1982). This contrasts to the hypoplasia, atrophy and/or necrosis seen in organs such as thymus, bone marrow and testicle which are not targets for carcinogenicity (Poland and Knutson 1982). 2,3,7,8-TCDD also causes a significant decrease in the spontaneous incidence of secretory gland tumors (Kociba, 1984). Furthermore, in genetically hairless mice (hr/hr) of the HRS/J strain, 2,3,7,8-TCDD produces hyperplasia and induces terminal differentiation of the epithelial keratinocytes. In mice of the same strain that are heterozygous for the hairless gene (hr/+) and which, therefore have normal hair, the skin is not sensitive to the promotional effects of 2,3,7,8-TCDD; nor does 2,3,7,8-TCDD produce a hyperplastic response in the skin.

It is clear, however, that induction of hyperplasia alone can not explain the high potency of 2,3,7,8-TCDD as a carcinogen. Many chemicals which produce as much hyperplasia as 2,3,7,8-TCDD are not carcinogenic. The tumor promoter TPA, for example, is a very strong inducer of hyperplasia in the skin, yet it is almost completely inactive as a complete carcinogen; and ethylphenylpropiolate which causes epidermal hyperplasia is not a promoter of skin carcinogenicity (Slaga 1983).

G.4.2 ROLE OF THE AH RECEPTOR

One effect of 2,3,7,8-TCDD that was recognized and investigated in experimental animals at an early stage was its activity in the induction of a number of enzymes involved in the metabolism of exogeneous chemicals, particularly in the liver. Of particular interest was the ability of 2,3,7,8-TCDD to induce cytochrome P-450 dependent enzymes. This group of enzymes, originally discovered in the liver, monoxygenate a broad spectrum of lipophilic substances. The carbon monoxide complex of these enzymes maximally absorbs light of about 450 mμ because of the presence of the heme group. These enzymes are found bound to membranes within cells (Whitlock 1986). They are "known to metabolize almost everything in the the Merck Index -- from small molecules such as ethanol . . . to large molecules such as 2,3,7,8-TCDD, benzo[a]pyrene and synthetic steroids. Virtually all drugs are substrates" (Ebert et al. 1983).

Poland and Glover (1973) investigated the ability of polychlorinated dibenzo-p-dioxin (PCDD) congeners to induce AHH activity in chicken embryos. They found a well-defined structure-activity relationship: 2,3,7,8-TCDD was the most active chlorinated congener in inducing AHH activity. Other congeners lacking chlorines in any one or more of the 2,3,7 and 8 positions or having chlorines in the 1,4,6, or 9 positions were less active or altogether inactive. This finding suggested that there was a strong steric requirement for induction of AHH activity as might be imposed by the need to bind to the active site of a receptor.

Concurrently Nebert and Gielen (1972) found that strains of mice differed in their ability to respond to the induction of AHH activity by PAHs and 2,3,7,8-TCDD and that the ability to respond was under genetic control. Together the two lines of evidence, i.e. structure-activity relationships and genetic segregation of AHH inducibility led Poland and Glover (1975) to postulate that enzyme induction by PAH and some halogenated aromatic hydrocarbons was mediated by a receptor. The receptor, when bound by 2,3,7,8-TCDD or another ligand, is hypothesized to bind to a particular region(s) of DNA and to "turn on" the genes for the cytochrome P-450 dependent enzymes. The unbound receptor is much less active or inactive. The putative receptor was designated the Ah receptor and the dominant allele for responsiveness was indicated by Ah^b and the recessive allele for nonresponsiveness by Ah^d (Nebert and Felton 1975 as cited by Eisen et al. 1983). Subsequent research indicated that the genes that code for cytochrome P-450 dependent enzymes were present in nonresponsive strains of mice but that some defect in the receptor either eliminated or reduced the affinity of the receptor for PAHs and 2,3,7,8-TCDD (Eisen et al. 1983).

2,3,7,8-TCDD is the most potent known inducer of AHH. In rat liver, 2,3,7,8-TCDD is 30,000 times more potent than 3-MC as an inducer. Moreover, 3-MC does not induce AHH in "non-responsive" mice strains. 2,3,7,8-TCDD, on the other hand, induces AHH in both responsive and non-responsive strains, albeit the non-responsive strains require a higher dose of 2,3,7,8-TCDD to achieve the same degree of induction. 2,3,7,8-TCDD is only very slowly metabolized by cytochrome P-450 dependent enzymes (Neal 1984), and, therefore, remains unaltered in the cell for long periods of time. Its resistance to metabolism probably contributes to its potency as an inducer.

G.4.3 THE AH RECEPTOR AND CANCER

The role of the Ah receptor in the carcinogenic activity of 2,3,7,8-TCDD is not clear. Poland and co-workers (Poland et al. 1983; Poland 1984) have shown that the promotion of skin papillomas by 2,3,7,8-TCDD in mice initiated with dimethylbenzanthracene segregates with the hr gene in HRS/J mice and that avidity of dioxins and furans for the Ah receptor parallels their promoter activity.

2,3,7,8-TCDD may promote carcinogenic activity by increasing cell proliferation and terminal differentiation or by suppressing immunologic responses that are important defense mechanisms against cancer cells. Both the proliferative effects and immunosuppressive effects of 2,3,7,8-TCDD appear to be mediated by a receptor. The exact involvement of the receptor in immunosuppression by dioxins and furans other than 2,3,7,8-TCDD may be congener-specific. Holsapple, McCay and Barnes (1986) exposed adult female mice to 2,3,7,8-TCDD, 2,7-dichlorodibenzo-p-dioxin (DCDD), or octachloro-dibenzo-p-dioxin (OCDD) for 14 days. Total doses were 2,3,7,8-TCDD, 1.0 µg/kg; DCDD 0.1, 1.0 or 10.0 µg/kg; OCDD 1.0 and 10.0 µg/kg. 2,3,7,8-TCDD produced strong immunosuppression. DCDD, which lacks affinity for the Ah receptor, caused comparable immunosuppression. OCDD, which also lack affinity for the receptor, caused no immunosuppression. Holsapple, McCay, and Barnes (1986) suggest that the immunosuppressive effects of DCDD might indicate that a receptor other than the Ah receptor is involved in its activity or that there is some direct interaction between DCDD and some subpopulations of cells involved in immunocompotence.

Taken together this evidence suggests that the carcinogenic effects of 2,3,7,8-TCDD may be mediated by the Ah receptor and that organisms that have high levels of receptor and demonstrate the full pleiotropic response to 2,3,7,8-TCDD might be expected to be more susceptible to the carcinogenic effects of 2,3,7,8-TCDD. Unfortunately, 2,3,7,8-TCDD has not been tested for its carcinogenic potential in "nonresponsive" mouse strains so direct evidence of genetic segregation of carcinogenic responsiveness is not available, and, of course, even if a correlation exists in mice, it may not extend to other species.

Okey et al. (1983) have summarized three pieces of evidence that suggest that the Ah receptor is not required for the expression of carcinogenesis by chemicals that bind to the receptor. First, some chemicals such as naphthoflavone bind to the Ah receptor with high affinity but are not carcinogenic. Second, both 3-MC and benzo(a)pyrene induce skin tumors in DBA/2J mice, a strain which is "nonresponsive" and, therefore, deficient in receptors. Finally 3-MC induces malignant transformation in C3H/10½ cells even though 3-MC does not bind to the Ah receptor or induce AHH cytochrome P-450 in these cells. The validity of the last piece of evidence has been called into question. Abernethy et al. (1985) found that 2,3,7,8-TCDD by itself did not induce transformation of cultured C3H/10T½ cells, but it did increase the number of transformed cells in a dose-related manner when the cells were first treated with the direct acting mutagen, MNNG (N-methyl-N-nitro-N-nitrosoguanidine). The authors concluded that 2,3,7,8-TCDD acted solely as a promoter in this assay system and that it was 100 to 10,000 times more potent that TPA.

At present, the mechanism by which 2,3,7,8-TCDD causes an increase in cancer in experimental animals is unknown. Some evidence suggests that it acts as a promoter. Furthermore, some less convincing evidence indicates that this effect may be mediated by binding to the Ah receptor.

G.4.4 IMMUNOSUPPRESSION

The possible role of immunosuppression in enhancing tumor incidence has been considered for many years. There is certainly much evidence that 2,3,7,8-TCDD causes immunosuppression in experimental animals; the picture is less clear for humans (see Dean and Kimbrough 1986). 2,3,7,8-TCDD causes atrophy of the thymus and, to a lesser extent, the peripheral lymph nodes in young animals (Thomas and Faith 1985). This is associated with impairment of thymus-dependent immune functions in rats (Faith and Moore 1977). At dose levels below those causing thymic atrophy, as low as 4 ng/kg, 2,3,7,8-TCDD suppresses the generation of cytotoxic T-lymphocytes (CTL) in lymph nodes and spleen in mice (Clark et al. 1981). This effect on CTL is apparently due to the induction by 2,3,7,8-TCDD of T-suppressor cells (Clark et al. 1981, 1983). 2,3,7,8-TCDD also suppresses production of antibodies to T-dependent and T-independent antigens in mice (Hinsdill et al. 1980; Vecchi et al. 1980)

Interestingly, the immunotoxic effects of 2,3,7,8-TCDD appear to be modulated by the Ah locus, since sensitivity to 2,3,7,8-TCDD's immunotoxic effects segregate with the Ah locus in "responsive" and "nonresponsive" strains of mice (Silkworth and Vecchi 1985). Immunosuppression in "nonresponsive" mice (D2 and AKR) required doses of 2,3,7,8-TCDD 25-times higher than in "responsive" mice (B6 and C3H/HeN). Also, the potency of 2,3,7,8-TCDD analogs as immunosuppressants correlates with their affinity for the Ah receptor (Silkworth and Vecchi 1985).

As noted above, the results of Holsapple, McCay, and Barnes (1986) suggest that other receptors or activities might also be involved in 2,3,7,8-TCDD-associated immunosuppression. At least, immunosuppression by DCDD appears not to involve the Ah receptor. It may be hypothesised that such an effect on the immune system could affect the incidence of cancer if cells at various stages of the development of cancer are normally subject to being killed by components of the immune system. As already described, there is some circumstantial evidence that this is true for some tumor types (Herberman 1985). However, the involvement of immunosuppression in the carcinogenicity of 2,3,7,8-TCDD has not been clearly demonstrated.

G.5 EXTRAPOLATION TO PCDD/PCDF

The evaluation of human health risks associated with exposure to a chemical for which toxicity in humans has not been directly measured must be based on data from studies involving other animal species. Due to limitations in the sensitivity (biological and statistical) of experimental procedures, and the relatively low levels of exposure to chemicals that humans generally experience, human risk assessment must also rely on inferences drawn from effects seen at high experimental dose levels to estimate risks at low dose levels typical of human exposure. These two types of extrapolations -- interspecies extrapolation and high-to-low dose extrapolation -- are typically performed in the course of evaluating the human health risks associated with exposures to many chemicals. The assessment of risk associated with exposure to polychlorodibenzo-p-dioxins (PCDD) and to polychlorodibenzofurans (PCDF) is further complicated by the fact that toxicity data for most forms of PCDD/PCDF are very limited. In most cases, data for 2,3,7,8-TCDD must be extrapolated to other PCDD/PCDF; this type of extrapolation is discussed below.

General conclusions about the mechanism of 2,3,7,3-TCDD toxicity can be extrapolated to other PCDDs and PCDFs based on a number of investigations of the activities of these chemicals in Ah receptor binding, enzyme induction, and toxicity. Poland, Glover, and Kende (1978) made the first investigation of the relative activity of various PCDDs and PCDFs in binding to the Ah receptor. This experiment showed that the number and location of chlorine atoms determined binding avidity and that PCDDs and PCDFs substituted with chlorine in the 2,3,7, and 8 positions bound more strongly than the others. The finding that each of the halogenated dibenzo-p-dioxins and furans competed with 2,3,7,8-TCDD for protein binding sites indicated that all PCDDs and PCDFs bind to the same receptors. Poland, Greenlee, and Kende (1979) demonstrated good correlations between the affinity of a PCDD or PCDF for the Ah receptor and its activity in induction of cytochrome P450-dependent enzymes in the chick. These early studies showed that various PCDDs and PCDFs bound to the Ah receptor and inducted P450 enzymes in a manner analogous to 2,3,7,8-TCDD. None was so efficient as 2,3,7,8-TCDD in causing these effects, and the efficiency of 2,3,7,8-substituted PCDDs and PCDFs was greater than that of the others. Insofar as receptor binding and enzyme induction related to toxicity, these studies are consistent with a common mechanism of toxicity for all PCDDs and PCDFs (Neal 1984).

Safe (1986; 1987) has studied correlations between the activity of PCDDs as inducers of cytochrome P450-dependent enzymes and their toxicity in rats. Those PCDDs and PCDFs that were most active as AHH inducers were also the most toxic as measured by weight loss and thymic atrophy. Safe (1987) made reference to unpublished experiments that show good correlations between enzyme induction and decreased body weight in guinea pigs and between enzyme induction and teratogenicity in mice.

Therefore, those PCDDs and PCDFs that are most potent as enzyme inducers also appear to be the most toxic; those less potent as inducers appear less toxic. Since the PCDD that are less toxic than 2,3,7,8-TCDD are also less active than 2,3,7,8-TCDD as carcinogens, it is reasonable to assume that there are general correlations among activities in enzyme induction, acute toxicity, and carcinogenicity.

The conclusion that the carcinogenicity mechanisms of all PCDDs and PCDFs are similar has not been directly demonstrated. It is possible that the carcinogenicity of the various PCDDs and PCDFs is not directly related to other effects, and works through mechanisms specific to each chemical. However, there are no data to support such a hypothesis, and it can be regarded as less tenable than that of a common mechanism. If the carcinogenic mechanism of

2,3,7,8-TCDD involves a threshold, then the carcinogenic mechanism of all other PCDDs and PCDFs is also likely to involve a threshold. On the other hand, if there is no threshold for 2,3,7,8-TCDD, there is likely to be none for the other PCDDs and PCDFs.

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APPENDIX H

ESTIMATION OF THE TOXIC POTENTIAL OF CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS

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APPENDIX H

ESTIMATION OF THE TOXIC POTENTIAL OF CHLORINATED DIBENZO-p-DIOXINS AND DIBENZOFURANS

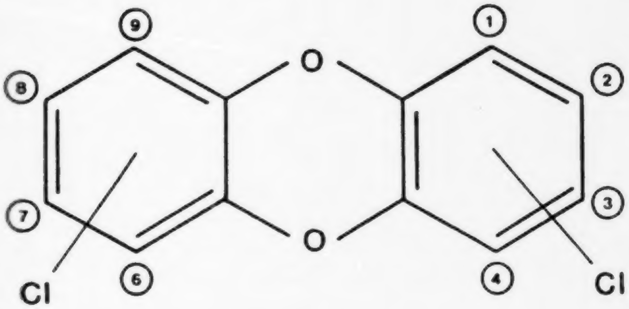
H.1 INTRODUCTION

A critical issue in performing the OWMC risk assessment is the evaluation of the toxic potential of two closely related families of complex chlorinated hydrocarbons, chlorinated dibenzo-p-dioxins (CDDs) and chlorinated dibenzofurans (CDFs). Certain forms of CDD and CDF are extremely toxic and have been identified as potent carcinogens in laboratory animals. In the Phase 4A Risk Assessment, risks associated with exposure to emissions from the proposed OWMC incinerator were driven by CDDs and CDFs, which may be formed during the combustion of hazardous wastes.

As shown in Figure H-1, CDDs and CDFs can contain between one and eight chlorine atoms. The individual forms, or congeners, of CDD and CDF vary in terms of the number and location of these chlorine atoms. There are a total of 75 congeners of CDD, and 135 congeners of CDF.

Figure H-1

Generalized Chemical Structure,
Chlorinated Dibenzo-p-Dioxins and Dibenzofurans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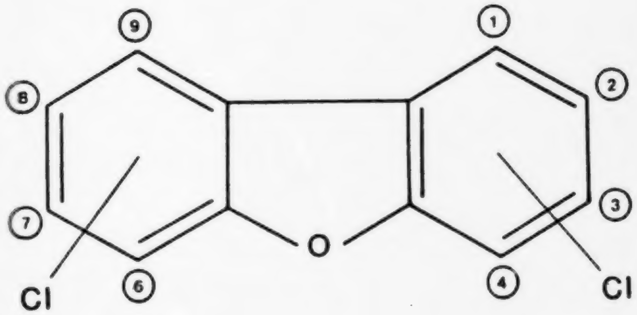
Chlorinated Dibenzo-p-dioxins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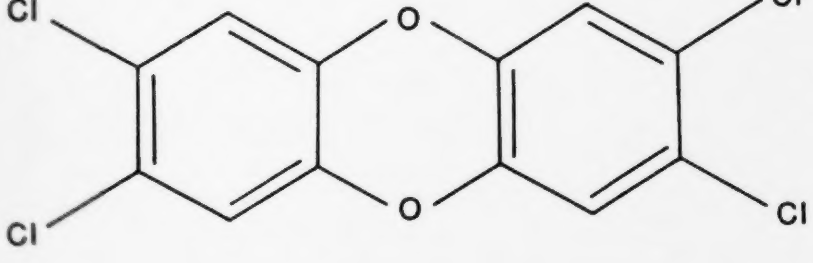
Chlorinated Dibenzofurans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2,3,7,8-Tetrachlorinated Dibenzo-p-dioxin

The various congeners of CDD and CDF are generally classified according to the number of chlorine atoms present. For example, CDDs with four chlorine atoms are referred to as tetrachlorinated dibenzo-p-dioxins (TCDD). Groups of CDDs and CDFs with the same number of chlorine atoms are known as homologs. Common abbreviations for the homologs class and the number of congeners in each homolog are summarized in Table H-1. Individual congeners within each homolog are typically identified by the location of the chlorine atoms. These locations are designated by the numbers 1 through 9. For example, as shown in Figure H-1, 2,3,7,8-TCDD has chlorine atoms in the 2, 3, 7, and 8 positions.

It is generally accepted that the toxicities of the various CDDs and CDFs vary according to both the number and location of chlorine atoms present. 2,3,7,8-TCDD, usually considered to be the most toxic of the CDDs and CDFs, is the most thoroughly studied. In contrast, far less information is available regarding the toxicity of the other 74 CDDs and the 135 CDFs.

In the Phase 4A Risk Assessment, ENVIRON assumed that all polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) containing four or more chlorine atoms were as toxic as 2,3,7,8-TCDD (CDDs and CDFs with fewer than four chlorine atoms were not considered). Given that 2,3,7,8-TCDD is 25 times more potent than the only other CDDs

TABLE H-1

THE HOMOLOGS OF CHLORINATED DIBENZO-p-DIOXIN AND CHLORINATED DIBENZOFURAN

Homolog	Abbreviation	Number of Congeners
Monochlorinated dibenzo-p-dioxin	MCDD	2
Monochlorinated dibenzofuran	MCDF	4
Dichlorinated dibenzo-p-dioxin	DCDD	10
Dichlorinated dibenzofuran	DCDF	16
Trichlorinated dibenzo-p-dioxin	TrCDD	14
Trichlorinated dibenzofuran	TrCDF	28
Tetrachlorinated dibenzo-p-dioxin	TCDD	22
Tetrachlorinated dibenzofuran	TCDF	38
Pentachlorinated dibenzo-p-dioxin	PnCDD	14
Pentachlorinated dibenzofuran	PnCDF	28
Hexachlorinated dibenzo-p-dioxin	HxCDD	10
Hexachlorinated dibenzofuran	HxCDF	16
Heptachlorinated dibenzo-p-dioxin	HpCDD	2
Heptachlorinated dibenzofuran	HpCDF	4
Octachlorinated dibenzo-p-dioxin	OCDD	1
Octachlorinated dibenzofuran	OCDF	1

tested for carcinogenicity to date and that it is believed to be the most toxic of the CDD and CDF congeners, this assumption likely overestimates the risk associated with exposure to mixtures of CDD/CDF. There are two alternatives to the conservative approach used by ENVIRON in the Phase 4A Risk Assessment:

1. Consider only those congeners for which cancer potency data are available and ignore the remainder of the CDDs and CDFs. This would limit the evaluation of risk to 2,3,7,8-TCDD and two 2,3,7,8-hexachlorinated dibenzo-p-dioxins (2,3,7,8-HxCDD), the only forms of CDD/CDF that have been demonstrated to produce cancer in laboratory animals. Since many other CDD/CDF congeners are suspected of having some carcinogenic potential, this approach may lead to an underestimation of risk.

2. Use all available toxicological data to estimate the carcinogenic potency of the various congeners of CDD and CDF relative to 2,3,7,8-TCDD. This approach necessitates the use of indirect measures of carcinogenicity to develop "Toxic Equivalence Factors" (TEFs) for those CDD/CDF congeners for which no cancer data are available.

ENVIRON has reviewed the approaches available to estimate the carcinogenic potency of CDDs and CDFs and has concluded that it is appropriate to use TEFs to refine the assumptions used in the Phase 4A Risk Assessment. The application of an appropriate TEF methodology allows for a more realistic, and yet still conservative, estimate of the risks associated with the proposed OWMC facility.

H.2 TOXIC EQUIVALENCE FACTORS

In the absence of a complete set of chronic toxicological data, various regulatory agencies and groups of scientists have developed TEFs for the estimation of potential impacts of mixtures of CDDs and CDFs on human health. The basis for TEF schemes are structure-activity relationships* and limited experimental data indicating that many CDDs and CDFs have toxic properties similar in kind, if not degree, to those of 2,3,7,8-TCDD. Therefore, the potencies of individual CDDs and CDFs can be compared to that of 2,3,7,8-TCDD, and relative toxicities estimated. These relative toxicities can then be used to calculate the overall toxicity of a mixture of CDDs and CDFs.

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* Structure-activity relationships are based on knowledge regarding the manner in which the structure of a compound (or part of a compound) influences the nature and magnitude of the toxicity of the substance.

For example, consider one gram of a hypothetical CDD/CDF mixture consisting of the following congeners:

20 wt% 2,3,7,8-TCDD
30 wt% 2,3,7,8-TCDF
50 wt% 1,3,4,7,9-PnCDD

If certain experimental data indicate that 2,3,7,8-TCDF is one order of magnitude (a factor of 10) less potent than 2,3,7,8-TCDD, and that 1,3,4,7,9-PnCDD is two orders of magnitude (a factor of 100) less potent than 2,3,7,8-TCDD, the following TEFs can be assigned:

	Relative Potency (TEF)
2,3,7,8-PCDD	1.0
2,3,7,8-TCDF	0.1
1,3,4,7,9-PnCDD	0.01

The mass of the hypothetical mixture in terms of 2,3,7,8-TCDD equivalents can then be calculated using the following formula:

$$Mte = Mm \sum_{i}^{n} (\omega_i)(TEF_i)$$

Where:

Mte = Mass of the mixture in 2,3,7,8-TCDD equivalents

Mm = Total mass of the mixture

ω_i = The weight fraction of congener i in the mixture

TEF_i = The toxic equivalence factor for congener i

n = The number of congeners in the mixture

For the hypothetical mixture being considered, the toxicity can be calculated as follows:

$$Mte = Mm \sum_{i}^{n} (\omega_i)\ (TEF_i)$$

$$Mte = (1.0g) \times [(0.20)\times(1.0) + (0.30) \times (0.1) + (0.50)\times(0.01)]$$

$$Mte = 0.235g\ 2,3,7,8\text{-TCDD equivalents}$$

Therefore, 1 gram of the mixture under consideration is estimated to be as potent as 0.235 g of 2,3,7,8-TCDD.

The basis for the development of a TEF scheme is the identification of data which can be used to estimate the potency of the various CDD and CDF congeners relative to 2,3,7,8-TCDD, (i.e. TEF_i values). When evaluating potential cancer risk, the most relevant data would obviously be those from carcinogenicity studies. However, to date only 2,3,7,8-TCDD, a mixture of 2,3,7,8-HxCDDs, and 2,7-dichlorodibenzo-p-dioxins (2,7-DCDD) have been tested for carcinogenic potency and therefore alternative data must be used to evaluate the potency of the remainder of the CDD and CDF congeners.

Scientists have used a variety of data to estimate the relative carcinogenic potency of the CDD and CDF congeners. Most TEF schemes are based on a consideration of some or all of the following types of information:

- The potency of CDDs and CDFs in studies of carcinogenicity involving exposed laboratory animals.
- The potency of CDDs and CDFs in studies of reproductive effects in exposed laboratory animals.
- Acute toxicity of CDDs and CDFs as measured in laboratory animals. Acute toxicity is commonly characterized by an LD_{50} value, or the dose of a chemical required to kill 50% of a group of exposed test animals.
- The interaction of CDDs and CDFs with a particular cellular protein called the Ah receptor. This interaction, which can be measured in cell extracts, is believed to be a critical step in producing toxic responses in exposed populations.
- Biological effects produced by the interaction between CDDs and CDFs with the Ah receptor. Both in vivo (tests involving live animals) and in vitro (tests using cultured cells or other systems that do not require live animals) experiments, such as enzyme induction, and cell keratinization tests, are used to measure the interaction.

The usefulness of these toxicity indicators as surrogates for carcinogenicity data is dependent on the degree to which a relationship is believed to exist between the effect measured and a carcinogenic response. For each of the indicators described above, the relationship between the effect measured and carcinogenicity is based on a generally accepted understanding of certain steps in the mechanism by which carcinogenic and some non-carcinogenic responses are produced by 2,3,7,8-TCDD (see Appendix B).

Specific mechanisms leading to toxic responses associated with exposure to CDDs and CDFs are not completely understood. However, it is generally accepted that an intracellular protein (the Ah receptor) plays an important role in the induction of a variety of toxic effects. In tests involving 2,3,7,8-TCDD, binding with the Ah receptor has been correlated with microsomal enzyme induction (Nebert and Given 1972), acute lethality (Neal et al. 1982), thymic involution (Poland and Glover 1980), induction of cleft palate (Poland and Glover 1980), induction of hepatic porphyria (Jones and Sweeney 1982), keratinization of XB cells in culture (Knutson and Poland 1980), induction of hyperkeratosis in mice (Poland and Knutson 1982), suppression of the antibody response to sheep red blood cells (Vecchi et al. 1983), suppression of cytotoxic T lymphocyte generation (Clark et al. 1983 and Nagarkatti et al. 1984), promotion of skin tumors in hairless mice (Poland et al. 1983), and promotion of tumors in initiated rat livers (Pitot et al. 1980). Taken as a whole, this body of evidence supports the conclusion that binding of 2,3,7,8-TCDD to the Ah receptor is a necessary initial step in triggering many, if not all, of the biological responses observed in animals exposed to 2,3,7,8-TCDD.

The understanding that binding of 2,3,7,8-TCDD to the Ah receptor is a critical step in producing a variety of toxic responses as well as cancer suggests that it may be possible to use data on toxic endpoints other than carcinogenicity to evaluate the relative potency of CDD and CDF congeners. In addition, direct measurement of the interaction between CDDs/CDFs and the Ah receptor can also form the basis for estimating relative toxic potency.

H.3 A REVIEW OF EXISTING TEF METHODOLOGIES

H.3.1 INTRODUCTION

ENVIRON has conducted a review of various TEF schemes that have been developed to estimate the relative cancer potency of the CDD/CDF congeners. The objective of this review was to identify the most appropriate TEF methodology for use in the OWMC risk assessment.

Relative toxicity methodologies have been developed by the Ontario Ministry of the Environment (OMOE 1985), the United States EPA (1981 1986), the State of California (Staff of the California Department of Health Services, 1986), the State of New York (Eadon et al. 1982), the German Federal Health Agency (as cited in Suter-Hofmann and Schlatter 1986), and the Swiss Government (1982). The characteristics of the USEPA (1981), California, New York State, Swiss, and German TEF schemes are outlined briefly below:

- USEPA, 1981
  In 1981, USEPA used a highly simplified technique for evaluating CDD/CDF toxic equivalency. 2,3,7,8-TCDD was assigned a value of 1 and all other TCDDs were assumed to be as potent as the 2,3,7,8 congener and the remaining CDDs and CDFs were not considered. This methodology is outdated and does not incorporate any data pertaining to the relative toxicity of the various CDD/CDF congeners.

- State of California, 1986
  The State of California assumes that all TCDD, TCDF, PnCDD, and PnCDF with chlorines in the 2,3,7,8 position

are as potent as 2,3,7,8-TCDD. All HxCDD, HxCDF, HpCDD, and HpCDF substituted with chlorine at the 2,3,7, and 8 positions are assumed to be approximately one-third as potent as 2,3,7,8-TCDD. All other congeners are assigned a toxic equivalence of 0. This system does not consider experimental data regarding specific congeners.

- New York State, 1982
  New York State has developed TEFs based solely on lethality data (LD_{50} data) for guinea pigs. The use of LD_{50} values ignores other, more relevant information such as teratogenicity data.

- Swiss Federal Office for Environmental Protection, 1982
  The Swiss method employs data based solely on enzyme induction assays, and, except for TCDD, assumes that each congener within a homolog of CDD/CDF has the same toxicity as the most potent member of the homolog. The Swiss system ignores other relevant data, including information on carcinogenicity and reproductive/teratogenic effects.

- German Federal Health Agency, 1984
  The German scheme is a refined approach which differentiates between the toxicities for 2,3,7,8- and non-2,3,7,8-substituted congeners within each homolog. Mon-, di-, tri-, and octa-chlorinated dibenzo-p-dioxins and dibenzofurans are assigned a TEF factor of 0.

In the Phase 4B Risk Assessment, two methodologies (OMOE 1985 and USEPA 1986) have been evaluated in detail. These two schemes are discussed and contrasted in this section.

H.3.2 THE OMOE (1985) AND USEPA (1986) TEF METHODOLOGIES

The TEF methodologies developed by OMOE (1985) and USEPA (1986) are refined approaches which incorporate currently available toxicity data and information on structure-activity relationships. These TEF schemes are presented in Table H-2.

TABLE H-2

OMOE AND USEPA TOXIC EQUIVALENCE FACTORS

	OMOE[1]	USEPA[2]
Chlorinated dibenzo-p-dioxins		
MCDDs	0.0001	0
DCDDs	0.001	0
TrCDDs	0.01	0
2,3,7,8-TCDDs	1.0	1.0
Non-2,3,7,8-TCDDs	0.01	0.01
2,3,7,8-PnCDDs	0.1	0.5
Non-2,3,7,8-PnCDDs	0.1	0.005
2,3,7,8-HxCDDs	0.1	0.04
Non-2,3,7,8-HxCDDs	0.1	0.0004
2,3,7,8-HpCDDs	0.01	0.001
Non-2,3,7,8-HpCDDs	0.01	0.00001
OCDDs	0.0001	0
Chlorinated dibenzofurans		
MCDFs	0.0001	0
DCDFs	0.001	0
TrCDFs	0.01	0
2,3,7,8-TCDFs	0.5	0.1
Non-2,3,7,8-TCDFs	0.5	0.001
2,3,7,8-PnCDFs	0.5	0.1
Non-2,3,7,8-PnCDFs	0.5	0.001
2,3,7,8-HxCDFs	0.1	0.01
Non-2,3,7,8-HxCDFs	0.1	0.0001
2,3,7,8-HpCDFs	0.01	0.0001
Non-2,3,7,8-HpCDFs	0.01	0.00001
OCDFs	0.0001	0

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[1] OMOE 1985

[2] USEPA 1986

Both methodologies are based primarily on a consideration of carcinogenicity, teratogenicity, and enzyme induction data. However, OMOE and USEPA differ in the interpretation of these data, as is reflected in the different TEFs for certain CDD/CDF congeners. For example, OMOE has assigned a TEF factor of 0.01 to TrCDDs and TrCDFs; USEPA assumes a factor of 0 for these congeners. In addition, OMOE assumes that the most potent of the CDD/CDF congeners are those that have chlorine substitution at three or four of the lateral (2,3,7, and 8) positions. In contrast, the USEPA does not consider those congeners substituted at three of the 2,3,7,8 positions to be as potent as those with all four positions chlorinated.

For all but one group of CDD/CDF (2,3,7,8-PnCDDs), OMOE assigns TEF factors greater than or equal to those developed by the USEPA. The OMOE TEF methodology will therefore predict a greater carcinogenic potency for most mixtures of CDD/CDF than will the USEPA methodology. This is illustrated in Table H-3, which compares the predicted 2,3,7,8-TCDD toxic equivalent (TEQ) emissions from the proposed OWMC incinerator based on the OMOE and USEPA TEF methodologies. A brief comparison of the two methodologies follows.

TABLE H-3

PROJECTED EMISSION RATES FOR POLYCHLORINATED DIBENZO-p-DIOXINS (PCDD) AND POLYCHLORINATED DIBENZOFURANS (PCDF)

	TEF Factor		Emission Projection[2] (g/s)	TEQ Emission Rate (g/s)[1]	
	EPA	OMOE		EPA	OMOE
PCDD					
Tetra-					
2378-	1.0	1.0	2.3E-10	2.3E-10	2.3E-10
non 2378-	0.01	0.01	4.1E-09	4.1E-11	4.1E-11
Penta-					
2378-	0.5	0.1	6.0E-10	3.0E-10	6.1E-11
non 2378-	0.005	0.1	5.4E-09	2.7E-11	5.4E-10
Hexa-	0.04	0.1	1.1E-08	4.5E-10	1.1E-09
Hepta-	0.001	0.01	1.3E-08	1.3E-11	1.3E-10
Octa-	0	0.0001	3.7E-08	0.0E+00	3.7E-12
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Subtotal			7.2E-08	1.1E-09	2.1E-09
PCDF					
Tetra-					
2378-	0.1	0.5	2.8E-09	2.8E-10	1.4E-09
non 2378-	0.001	0.5	3.3E-08	3.3E-11	1.6E-08
Penta-					
2378-	0.1	0.5	6.4E-09	6.4E-10	3.2E-09
non 2378-	0.001	0.5	6.1E-08	6.1E-11	3.1E-08
Hexa-	0.01	0.1	9.9E-08	9.9E-10	9.9E-09
Hepta-	0.001	0.01	7.3E-08	7.3E-11	7.3E-10
Octa-	0	0.0001	4.5E-08	0.0E+00	4.5E-12
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Subtotal			3.2E-07	2.1E-09	6.2E-08
TOTAL			3.9E-07	3.1E-09	6.4E-08

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[1] Emission rates converted to 2,3,7,8-TCDD equivalents (TEQ).

[2] Emission data from NUKEM HIM Report - June, 1985.
Recommended for Phase 4B Risk Assessment (ORF 1987).
Gas conditions assumed reported as dry, 11.6% oxygen and converted to dry, 7% oxygen.
Based on 22860 Nm^3/hr (dry, 7% oxygen) 150 000 t/a facility, operational 7 000 hr/a.

H.3.2.1 Chlorinated Dibenzo-p-Dioxins

Monochlorinated Dibenzo-p-Dioxins (MCDDs). OMOE assigns MCDDs a TEF of 0.0001 based on studies indicating that the teratogenic potency of these compounds is at least four orders of magnitude less than that of 2,3,7,8-TCDD. The assignment of a non-zero TEF for MCDDs appears to incorporate health-protective considerations. There were no observed teratogenic effects in a test of MCDD; therefore the NOEL might be significantly higher than the highest dose tested. The USEPA does not explicitly consider MCDD in its equivalency scheme.

Dichlorinated Dibenzo-p-Dioxins (DCDDs). OMOE assigns DCDDs a TEF of 0.001 based on a test indicating that 2,7-DCDD "was apparently teratogenic" at a dose about 4 000 times greater than the teratogenic dose of 2,3,7,8-TCDD. At the same dose 2,3-DCDD did not exhibit teratogenicity. It should also be noted that a second test of 2,7-DCDD at a dose 200 000 to 400 000 times higher than the teratogenic dose of 2,3,7,8-TCDD did not produce a teratogenic response. These data suggest a variation in the teratogenic potency among the DCDDs which may not be captured in assigning a single TEF of 0.001. In addition, OMOE assesses a carcinogenicity test of 2,7-DCDD as demonstrating a suggestive effect. If 2,7-DCDD is carcinogenic, it is approximately 14 000 times less potent than

2,3,7,8-TCDD. The USEPA does not explicitly consider DCDD in its toxic equivalency scheme.

Trichlorinated Dibenzo-p-Dioxins (TrCDDs). OMOE assigns TrCDDs a TEF of 0.01 based on enzyme induction tests. The LD_{50} for 2,3,7-TrCDD is approximately 15 000 greater than the LD_{50} of 2,3,7,8-TCDD. Furthermore, although OMOE states that *in vitro* AHH induction by 2,3,7-TrCDD is from 157-fold to 2 750-fold less than that by 2,3,7,8-TCDD (p. 3 - 124), tabular data on *in vitro* AHH induction (Table 3.6.2 a) are presented only for the less potent finding. The assignment of a TEF of 0.01 appears to be conservative since both the LD_{50} and AHH induction data indicate that 2,3,7-TrCDD is less than 1/1,000 as potent as 2,3,7,8-TCDD. There are no data concerning the potential carcinogenicity or teratogenicity of these compounds. Unlike OMOE, USEPA does not consider the MCDD, DCDD and TrCDDs separately. Instead, it describes the potencies of all three congeners together and does not assign a value to any of them in its TEF scheme. No reason is given for excluding the MCDDs, DCDDs, and TrCDDs, but EPA states, "the toxicity data... do not strongly support this extended range of concern" (to include CDDs and CDFs with three chlorines in the 2,3,7, or 8 positions).

Tetrachlorinated Dibenzo-p-Dioxins (TCDDs) Both OMOE and USEPA assign 2,3,7,8-TCDD a TEF of 1.0. In addition, on the basis of enzyme induction, both OMOE and USEPA assign non-2,3,7,8-TCDDs a TEF of 0.01. This is the only homolog for which OMOE differentiates the toxicity of 2,3,7,8-substituted isomers from the non-2,3,7,8 substituted isomers. EPA makes that discrimination for all homologs.

Pentachlorinated Dibenzo-p-Dioxins (PnCDDs). OMOE assigns all PnCDDs a TEF of 0.1 based on enzyme induction. USEPA assigns 2,3,7,8-PnCDD a value of 0.5, which is the arithmetic mean of the relative carcinogenic potency values of 2,3,7,8-TCDD and HxCDD. Data on receptor binding, cell keratinization, and enzyme induction support this value. USEPA further assigns non-2,3,7,8-PnCDDs a value of 0.005 since non-2,3,7,8 congeners appear to be one to three orders of magnitude less toxic than respective 2,3,7,8 congeners based on *in vitro* test results.

Hexachlorinated Dibenzo-p-Dioxins (HxCDDs). OMOE assigns all HxCDD a TEF value of 0.1 based on teratogenicity and enzyme induction studies. USEPA assigns 2,3,7,8-HxCDD a TEF of 0.04 based on the tested carcinogenic potency of a mixture of two isomers relative to 2,3,7,8-TCDD. USEPA further assigns non-2,3,7,8-HxCDD a TEF of 0.0004 since non-2,3,7,8 congeners appear to be one to three orders of magnitude less toxic than 2,3,7,8 congeners in *in vitro* tests.

Heptachlorinated Dibenzo-p-Dioxins (HpCDDs). OMOE assigns all HpCDD a value of 0.01 based on enzyme induction and lethality (LD_{50}) data. USEPA assigns 2,3,7,8-HpCDD a value of 0.001 and non-2,3,7,8-HpCDD a value 0.00001 based on enzyme induction test results. USEPA does not consider LD_{50} data in developing TEFs, and OMOE states that enzyme induction by HpCDDs is 500-fold less than with 2,3,7,8-TCDD. The difference between the USEPA and OMOE TEFs for 2,3,7,8-HpCDDs apparently results from the fact that OMOE considers LD_{50} data, and treats available enzyme induction data differently than does the USEPA.

Octachlorinated Dibenzo-p-Dioxin (OCDD). OMOE assigns OCDDs a TEF value of 0.0001 based on enzyme induction. This assignment focuses on the most sensitive measure of OCDD toxicity. OCDD is more than 3 000 000 times less lethal and 500 000 times less teratogenic than 2,3,7,8-TCDD. USEPA assigns OCDDs a TEF value of 0.

H.3.2.2 Chlorinated Dibenzofurans

Monochlorinated Dibenzofurans (MCDFs). OMOE assigns MCDFs a TEF of 0.0001, stating that these isomers have toxicity equal to or less than OCDDs. One MCDF tested is 1 000 000-fold less active than 2,3,7,8-TCDD as an AHH inducer. The USEPA does not explicitly consider MCDF in its equivalency scheme.

Dichlorinated Dibenzofurans (DCDFs). OMOE assigns DCDFs a TEF of 0.0001. As with the MCDFs, OMOE states that the toxicity of the DCDFs is less than or equal to OCDD. The only measured effect of DCDFs (AHH induction) shows it to be less than 0.0001 as active as 2,3,7,8-TCDD. The USEPA does not explicitly consider DCDFs in its equivalency scheme.

Trichlorinated Dibenzofurans (TrCDFs). By analogy with the TrCDDs, OMOE assigns a TEF of 0.01 to all TrCDFs. The only measured effect for a TrCDF (AHH induction), shows it to be 1000 times less active than 2,3,7,8-TCDD. The USEPA does not explicitly consider TrCDFs in its equivalency scheme.

Tetrachlorinated Dibenzofurans (TCDFs). OMOE assigns all TCDFs a TEF of 0.5 based on enzyme induction and lethality (LD_{50}). This TEF value is based on data that indicate that 2,3,7,8-TCDF is 1/3 as toxic measured by LD50 and about 60% as active as 2,3,7,8-TCDD in inducing AHH.

USEPA assigns 2,3,7,8-TCDF a TEF of 0.1 based on data on reproductive effects, enzyme induction, and receptor binding studies. Reproductive studies indicate that the toxicity of 2,3,7,8-TCDF is approximately 10 times less than that of 2,3,7,8-TCDD. USEPA further assigns non-2,3,7,8-TCDF a TEF of 0.001 based on *in vitro* test results which indicate that non-2,3,7,8 congeners are approximately one to three orders of magnitude less active in enzyme induction tests than 2,3,7,8 congeners.

Pentachlorinated Dibenzofuran (PnCDFs). OMOE assigns all PnCDFs a TEF value of 0.5 based on enzyme induction studies that showed a PnCDF congener was 30% as active as 2,3,7,8-TCDD. Other PnCDFs tested were less active. USEPA assigns 2,3,7,8-PnCDFs a value of 0.1 based on enzyme induction. Evidently, USEPA's evaluation of enzyme induction data weighed the evidence that some 2,3,7,8 substituted PnCDF congeners induce enzymes with less than 30% of the activity of 2,3,7,8-TCDD. USEPA assigns non-2,3,7,8 PnCDFs a value of 0.001 since these congeners are approximately one to three orders of magnitude less active than in *in vitro* tests 2,3,7,8-PnCDFs.

Hexachlorinated Dibenzofuran (HxCDFs). OMOE assigns all HxCDFs a TEF value of 0.1 based on enzyme induction and lethality (LD_{50}) data, both of which indicate that HxCDFs are about 60 fold less active than 2,3,7,8-TCDD. USEPA assigns 2,3,7,8-HxCDF a value of 0.01 based on enzyme induction studies that show 2,3,7,8-HxCDFs are 10 times less active than P_nCDFs. Non-2,3,7,8-HxCDF are assigned a value of 0.0001 based on *in vitro* data. Again, the difference between OMOE and USEPA may result from different approaches to rounding the data. According to OMOE's table of enzyme induction data, none of the HxCDFs caused significant induction. However, the non-significant induction data were apparently weighed more heavily by OMOE than by USEPA.

Heptachlorinated Dibenzofuran (HpCDFs). OMOE assigns all HpCDFs a value of 0.01; USEPA assigns 2,3,7,8-HpCDF a value of 0.001 and other HpCDFs a value of 0.0001. USEPA relied on limited *in vitro* test results whereas OMOE, in this case, based its evaluation on analogy to HpCDDs.

Octachlorinated Dibenzofuran (OCDF). OMOE comments that there are no available data for OCDF and assigns it a TEF of 0.0001, the same value as for OCDD. USEPA, in parallel with its decision regarding OCDD, assigns a TEF of 0 to OCDF.

H.3.2.3 Summary

There are two significant differences between the USEPA and OMOE toxic equivalence factor methodologies:

- With the exception of TCDD, OMOE assigns the same TEFs to all isomers within a homolog. USEPA considers all non-2,3,7,8 isomers to be 100 times less potent than 2,3,7,8 isomers.
- OMOE assigns TEFs >0 to mono-, di-, tri-, and octa-CDDs and CDFs. USEPA assigns TEFs of 0 to all members of the mono-, di-, tri-, and octa- homologs.

In general, ENVIRON believes that the USEPA scheme is the most appropriately sound methodology. For example, with regard to the relative TEFs of 2,3,7,8 and non-2,3,7,8 isomers within each homolog, ENVIRON concludes that the USEPA method makes better use of the available data. In every case where information is available, the non-2,3,7,8 isomers are less active. The use of a factor of 0.01 by the USEPA to reduce the toxicity of those isomers is consistent with the observed 10 to 1 000-fold difference in measurements between 2,3,7,8 and non-2,3,7,8 isomers.

One reason for OMOE's favoring a single TEF for each homolog was the difficulty of measuring amounts of different isomers in each homolog. Studies by USEPA (1986) and others have demonstrated that isomer distributions in incinerator fly ash samples tend to be uniform, and that methods for estimating the amounts of individual congeners are available. Therefore, analytical limitations do not appear to exclude the use of different TEFs for 2,3,7,8 and non-2,3,7,8 isomers.

Measurements of mono-, di-, and tri- isomers are not routinely made. Therefore, only limited use can be made of the values assigned to these compounds by OMOE. If those measurements become routine, the OMOE TEFs will take on more significance. A review of the OMOE document indicates that the TEFs chosen by that organization for mono-, di-, and tri- isomers are sufficiently health protective. In the case of OCDDs and CDFs, the assignment of 0.0001 by OMOE was based on the most sensitive measure of toxicity for OCDD. Other measures would indicate that OCDD is much less toxic, and would therefore be assigned a TEF factor much lower than 0.0001. In any case, OCDD/OCDF would not contribute significantly to the toxicity of the mixture predicted to be emitted from the proposed OWMC incinerator.

H.4 APPLICATION OF TOXIC EQUIVALENCE SCHEMES

H.4.1 INTRODUCTION

Toxic equivalence schemes have been applied worldwide to estimate the toxicity of incinerator emissions and other materials contaminated with mixtures of CDD/CDF. The accuracy of these schemes has been evaluated by comparing the toxicity predicted by various methodologies with measured toxicities. Studies performed to date indicate that the OMOE toxic equivalency scheme predicts a greater toxic effect than the EPA scheme, and that based on animal toxicity, the EPA method has produced more accurate estimates in the few cases examined. Studies providing supporting evidence for this are summarized below.

H.4.2 INCINERATOR FLYASH (EPA)

USEPA (1986) applied its TEF methodology to fly ash samples collected from two municipal solid waste incinerators. As shown on Table H-4, using the EPA TEF methodology, one sample (sample A) had a total of 38 2,3,7,8-TCDD equivalents (TEQ) when all members of each homolog were assumed to be as toxic as the 2,3,7,8 isomers. When proportions of 2,3,7,8 to total isomers were considered, sample A was calculated to contain 2 TEQ. In the second sample (sample B) the TEQ totals were 9 and 0.7, respectively.

In comparison, as shown in Table H-5, application of the OMOE TEFs to sample A produced estimates of 42.6 TEQ when all isomers were considered to be as toxic as the 2,3,7,8 isomers and 30.4 TEQ when 2,3,7,8-TCDD was considered to be 100 times more toxic than the other TCDDs. For sample B, the TEQ totals were 19.4 and 17.2, respectively.

Measured values for aryl hydrocarbon hydroxylase (AHH) induction, ethoxyresorufin O-deethylase (EROD) induction, and receptor binding are presented for the two flyash samples in Tables C-4 and C-5. For both samples, the EPA and OMOE TEF schemes predict greater toxicity than measured by the AHH and EROD assays if all isomers are assumed to be substituted in the 2,3,7 and 8 positions. The EPA scheme, however, underestimates receptor binding for both samples, while the OMOE scheme

TABLE H-4

APPLICATION OF THE USEPA TEF METHODOLOGY TO FLYASH FROM AN MSW INCINERATION FACILITY[1]

			Sample A			Sample B		
Isomer	TEF	Propn. Factor (PF)	Conc. (ppb)	TEQ (ppb) W/O PF	TEQ (ppb) W/PF	Conc. (ppb)	TEQ (ppb) W/O PF	TEQ (ppb) W/PF
Total TCDDs	1	1	12.9	12.9		2.4	2.4	
2,3,7,8-TCDDs	1	0.05	12.9		0.6	2.4		0.1
Other TCDDs	0.01	0.95	12.9		0.1	2.4		--
Total P_nCDDs	0.5	1	37.5	18.8		7.9	4.0	
2,3,7,8-P_nCDDs	0.5	0.07	37.5		1.3	7.9		0.3
Other P_nCDDs	0.005	0.93	37.5		0.2	7.9		--
Total H_xCDDs	0.04	1	75	3		9.7	0.4	
2,3,7,8-H_xCDDs	0.04	0.3	75		0.9	9.7		0.1
Other H_xCDDs	0.0004	0.7	75		--	9.7		--
Total H_pCDDs	0.001	1	41.9	--		9.1	--	
2,3,7,8-H_pCDDs	0.001	0.5	41.9		--	9.1		--
Other H_pCDDs	0.00001	0.5	41.9		--	9.1		--
Total TCDFs	0.1	1	8.2	0.8		4.4	0.4	
2,3,7,8-TCDFs	0.1	0.03	8.2		--	4.4		--
Other TCDFs	0.001	0.97	8.2		--	4.4		--
Total P_nCDFs	0.1	1	19.8	2.0		21.0	2.1	
2,3,7,8-P_nCDFs	0.1	0.07	19.8		0.1	21.0		0.1
Other P_nCDFs	0.001	0.93	19.8		--	21.0		--
Total H_xCDFs	0.01	1	38.7	0.4		21.6	0.2	
2,3,7,8-H_xCDFs	0.01	0.25	38.7		0.1	21.6		0.1
Other H_xCDFs	0.0001	0.75	38.7		--	21.6		--
Total H_pCDFs	0.001	1	20.6	--		16.6	--	
2,3,7,8-H_pCDFs	0.001	0.50	20.6		--	16.6		--
Other H_pCDFs	0.00001	0.50	20.6		--	16.6		--
Total TEQ				38	3.3		9	0.7
Measured TEQ								
	AHH bioassay				4			2
	EROD bioassay				5			2
	Receptor binding assay				62			11

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[1] Adapted from USEPA 1986.

TABLE H-5

APPLICATION OF THE OMOE TEF METHODOLOGY TO FLYASH FROM AN MSW INCINERATION FACILITY

Isomer	TEF	Propn. Factor	Sample A Conc. (ppb)	Sample A TEQ (ppb) A	Sample A TEQ (ppb) B	Sample B Conc. (ppb)	Sample B TEQ (ppb) Ac	Sample B TEQ (ppb) Bc
Total TCDDs	1	1	12.9	12.9		2.4	2.4	
2,3,7,8-TCDDs	1	0.05			0.6			0.12
Other TCDDs	0.01	0.95			0.1			0.023
Total P_nCDDs	0.1	1	37.5	3.8	3.8	7.9	0.8	0.8
Total H_xCDDs	0.1	1	75	7.5	7.8	9.7	1.0	1.0
Total H_pCDDs	0.01	1	41.9	0.4	0.4	9.1	0.1	0.1
Total TCDFs	0.5	1	8.2	4.1	4.1	4.4	2.2	2.2
Total P_nCDFs	0.5	1	19.8	9.9	9.9	21.0	10.5	10.5
Total H_xCDFs	0.1	1	38.7	3.8	3.8	21.6	2.2	2.2
Total H_pCDFs	0.01	1	20.6	0.2	0.2	16.6	0.2	0.2
Total TEQ				42.6	30.4		19.4	17.02
Measured TEQ								
AHH bioassay					4			2
EROD bioassay					5			2
Receptor binding assay					65			11

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[1] Adapted from USEPA 1986.

underestimates receptor binding for only one sample. EPA believes that receptor binding demonstrates a poorer correlation with toxicity than the enzyme induction assay and, therefore, is not as accurate a measure of toxicity as the AHH and EROD assays. When a breakdown between 2,3,7,8 and non-2,3,7,8 congeners is assumed, enzyme induction is slightly underpredicted using the scheme developed by USEPA. The OMOE scheme, on the other hand, significantly over-predicts enzyme induction even when 2,3,7,8 and non-2,3,7,8 congeners are considered separately. Both TEF schemes appear to be protective of human health, with the OMOE methodology predicting greater toxicity than that of the USEPA.

H.4.3 INCINERATOR FLYASH (Suter-Hofmann and Schlatter)

Suter-Hofmann and Schlatter (1986) have measured the potency of municipal incinerator flyash as a systemic poison in laboratory rats. In the study, flyash was analyzed for CDDs and CDFs, and three TEF schemes (Swiss, Eadon, and German) were used to estimate 2,3,7,8-TCDD equivalents (TEQ). They then tested metal-free flyash extract in the rats since the toxicity of lead and cadmium was so great that it limited the amount of unfractionated flyash that could be fed to rats.

The greatest quantity of flyash extract fed to rats in the Suter-Hofmann and Schlatter study corresponds to 288, 1 560, and

1 800 ng/kg/d TEQs using the German, Swiss, and Eadon TEF schemes, respectively. That quantity of extract caused changes in thymus, liver, and total body weights that were comparable to those observed by Kociba et al. at a dose of 100 ng/kg/d (1978). Based on that comparison, Suter-Hofmann and Schlatter concluded that the German TEF scheme most accurately predicted the toxicity of the PCDD/PCDF mixture; their results showed that the Swiss and Eadon methodologies produced a 10 to 20 fold overestimate of toxicity.

ENVIRON applied the OMOE and USEPA schemes to calculate TEQs from the data presented in the Suter-Hofmann and Schlatter paper. As can be seen in Table H-6, the OMOE scheme produces a larger estimate than any other scheme. The USEPA scheme agrees within a factor of 1.3 with the German scheme, which Suter-Hofmann and Schlatter concluded "gave reasonable predictions."

All of the TEF schemes are predicated on the existence of some relationship between acute effects (enzyme induction and receptor binding) and chronic effects, e.g. carcinogenicity. If the same relationship that holds between acute and chronic toxicity for 2,3,7,8-TCDD also applies to mixtures of dioxins and furans, then the EPA and German schemes would appear to be more accurate than the OMOE, Swiss, and Eadon methodologies.

H.4.4 CONCLUSIONS

The data presented in Tables H-4, H-5, and H-6 differ in the endpoints examined. In the first two tables, TEQs are compared to enzyme induction data; in Table H-6, calculated TEQs are compared to whole animal toxicity data. The precise relationships between any of these data and carcinogenicity remain unclear. However, based on available information, both the USEPA and OMOE TEF methodologies appear to be health protective. The higher protection factor associated with the OMOE scheme is achieved largely by assigning single TEF values to entire homologs of CDDs and CDFs. Since methods are available to measure or estimate specific congener concentrations, there is now no overriding reason to apply a single factor to an entire homolog. Therefore, the EPA method, which considers the lower toxicity of the non-2,3,7,8 congeners and appears to predict toxicity reasonably well, is considered to be suitable for estimating TCDD equivalents.

TABLE H-6

COMPARISON OF USEPA AND OMOE TOXIC EQUIVALENCE METHODOLOGIES USING DATA FROM SUTER-HOFMANN AND SCHLATTER[1]

	OMOE			EPA		
	Amt	TEF	TEQ	Amt	TEF	TEQ
Tetra CDD	9.2	0.01	0.09	9.2	0.01	0.09
2,3,7,8	1.3	1.0	1.3	1.3	1.0	1.3
Penta CDD	48.2	0.1	4.8	48.2	0.005	0.24
2,3,7,8	6.3	0.1	0.6	6.3	0.5	3.2
Hexa CDD	67.5	0.1	6.7	67.5	0.004	0.3
2,3,7,8	12.7	0.1	1.3	12.7	0.04	0.51
Hepta CDD	8.5	0.01	0.08	8.5	0.00001	
2,3,7,8	17.3	0.01	0.17	17.3	0.001	0.02
OCDD	448	0.0001	0.04	448	0	0
Tetra CDF	313	0.5	156	303.6	0.001	0.3
2,3,7,8				9.4	0.1	0.94
Penta	220	0.5	110	204.6	0.001	0.20
2,3,7,8				15.4	0.1	1.54
Hexa	108	0.1	10.8	81	0.001	0.08
2,3,7,8				27	0.01	0.27
Hepta	137	0.01	1.4	68	0.0001	0
2,3,7,8				68	0.001	0
OCDF	310	0.0001	0.03	310	0	0
			293.3			9.0

Other TEF Scheme Results:

Swiss	65
Eadon	75
German	12

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[1] Suter-Hofmann and Schlatter, 1986.

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APPENDIX I

ESTIMATION OF HUMAN EXPOSURES TO CONTAMINANTS IN THE ENVIRONMENT

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APPENDIX I

ESTIMATION OF HUMAN EXPOSURES TO CONTAMINANTS IN THE ENVIRONMENT

I.1 INTRODUCTION

Exposures to contaminants released from the proposed OWMC special waste treatment and disposal facility can occur through a variety of pathways. For example, contaminants released into the atmosphere will be dispersed in the air and may be inhaled. Alternatively, the contaminants may deposit onto the ground, plants, buildings, or other surfaces. Individuals may be exposed to the deposited chemicals through ingestion of the plants or soil, or through dermal contact with contaminated surfaces. A variety of wildlife and livestock may also be exposed to facility emissions; consumption of meat or other products (e.g., milk and eggs) taken from these animals may also lead to human exposures.

It was not feasible to quantitatively evaluate each human exposure pathway that may be associated with the operation of the proposed OWMC facility. In the Phase 4B risk assessment, only those pathways believed to pose the greatest risk to human health or those specifically identified as a public concern were evaluated. In addition, it was necessary to restrict the assessment to those pathways of concern for which sufficient data were available to quantitatively assess human health risks.

Contaminant concentrations in the environmental media relevant to each exposure pathway examined were calculated using the approaches presented in Appendices D and E. This appendix describes the methods used in the Phase 4B risk assessment to estimate the magnitude of potential human exposures to contaminants in these media.

I.2 ROUTINE RELEASES TO THE ATMOSPHERE

I.2.1 INTRODUCTION

Three types of routine atmospheric emissions have been evaluated in the Phase 4B risk assessment: (1) emissions from the incinerator stack; (2) emissions from the evaporation unit of the physical/chemical plant; and (3) fugitive releases from various waste handling, storage and treatment operations. In the Phase 4B risk assessment, public health risks associated with routine atmospheric releases from the proposed OWMC facility have been assessed through an examination of the following exposure pathways:

- Direct inhalation.
- Ingestion of water taken from ponds and cisterns;
- Consumption of various fruits and vegetables;
- Consumption of beef, pork, chicken, and eggs;
- Consumption of milk;
- Incidental ingestion of soil; and
- Dermal contact with soil.

The formulae used to calculate potential human exposures for each of these pathways are presented in this section following a summary discussion of the major assumptions made in the assessment.

I.2.2 MAJOR ASSUMPTIONS IN THE ESTIMATION OF HUMAN EXPOSURES

The magnitude of exposure to contaminants released from the OWMC facility is dependent upon an individual's location, diet and behavior. For example, contaminants emitted from the incinerator stack will be dispersed in the atmosphere, and the highest annual average chemical concentrations in air will occur approximately one to two kilometers northeast of the facility. A person located at this point is likely to inhale a greater quantity of a given contaminant released from the incinerator than someone situated elsewhere in the site vicinity. Furthermore, since higher contaminant doses will occur the longer a person is exposed to the stack emissions, individuals located at different locations in the site vicinity for different periods of time would not be expected to receive the same dose of a chemical emitted from the incinerator stack.

As a result of uncertainties in characterizing the current and future behavior patterns of the human population in the site vicinity, it is difficult to estimate the likely levels of exposure for particular groups of individuals. To offset these uncertainties, conservative assumptions are typically made, and may result in an overestimate in the magnitude of human exposure, and subsequently of human health risk. In the Phase 4B risk assessment, the following conservative assumptions were made in predicting the magnitude of potential human exposures.

- For exposures via inhalation, all dose estimates were based on a person located permanently at the point of highest ground-level contaminant concentrations.
- For exposures resulting from ingestion of (or dermal contact with) soil, all dose estimates were based on a person permanently located at the point of highest ground-level contaminant concentrations. 100 percent of the soil ingestion and contact was assumed to occur at the location of maximum contamination.
- For exposures resulting from ingestion of crops, all dose estimates were based on the consumption of produce grown at the location of highest ground-level contaminant concentrations. 100 percent of the produce consumed by a person was assumed to be taken from the location of maximum contamination.
- For exposures resulting from ingestion of meat, milk, and eggs, all dose estimates are based on the consumption of livestock and poultry raised at the point of highest contaminant concentrations. Fodder for these animals was assumed to be taken from the

same point; water was assumed to be taken from the Welland River which in general had higher chemical concentrations than other bodies of surface water. 100 percent of the beef, pork, poultry, milk, and eggs consumed by a person was assumed to be taken from the location of maximum contamination.

- For exposures resulting from the ingestion of surface water, all dose estimates were based on the consumption of water taken from a pond or cistern located at the point of highest ground-level contaminant concentrations. 100 percent of the water ingested by the person was assumed to be taken from a pond or cistern located at the point of maximum contamination.
- All long-term exposures were assumed to occur continuously for a period of 70 years.
- The absorption rate for all chemicals inhaled or ingested was assumed to be 100% except as noted in Chapter 9.

In evaluating most pathways, a total lifetime dose was calculated assuming a body weight of 70 kg for the exposed individual. This value approximates the average weight of a man over the course of a 70-year lifetime (NIOSH 1980). For certain pathways, such as soil ingestion, potential exposures were calculated separately for children and adults, since children may exhibit significantly different patterns of behavior and thus exposure.

I.2.3 INHALATION

Long-term exposures through inhalation of air contaminated by facility emissions were calculated in the risk assessment using the following equation:

D_i = $(C_a)(I_a)(AF)/[(BW)(1000)]$

Where:

D_i = Dose via inhalation, mg/(kg-d)

C_a = Contaminant concentration in air, $\mu g/m^3$

I_a = Inhalation rate, m^3/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

It has been assumed in the risk assessment that the average breathing rate over the lifetime exposure will be 23 m^3/d (Altman and Dittmer 1974). This rate corresponds to an average man performing light work for 10 hours per day and resting 14 hours per day.

I.2.4 INGESTION OF BEEF

Long-term exposures through ingestion of beef from cattle raised in the vicinity of the proposed facility were calculated using the following equation:

D_b = $(C_b)(I_b)(AF)/[(BW)(1000)]$

Where:

D_b = Dose via ingestion of beef, mg/(kg-d)

C_b = Contaminant concentration in beef, $\mu g/kg$

I_b = Ingestion rate, beef, kg/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

It was assumed in the risk assessment that the total daily intake of beef (I_b) is 0.106 kg/d (Statistics Canada 1986). This value is based on the average rate of consumption of beef for the total Canadian population.

I.2.5 INGESTION OF PORK

Long-term exposures through ingestion of pork from pigs raised in the vicinity of the proposed facility were calculated using the following equation:

$$D_p = (C_p)(I_p)(AF)/[(BW)(1000)]$$

Where:

D_p = Dose via ingestion of pork, mg/(kg-d)
C_p = Contaminant concentration in pork, µg/kg
I_p = Ingestion rate, pork, kg/d
AF = Absorption factor, dimensionless
BW = Body weight, kg

It was assumed in the risk assessment that the total daily intake of pork, (I_p) is 0.078 kg/d (Statistics Canada 1986). This value is based on the average rate of consumption of pork for the total Canadian population.

I.2.6 INGESTION OF POULTRY

Long-term exposures through ingestion of poultry raised in the vicinity of the proposed facility were calculated using the following equation:

$$D_t = (C_t)(I_t)(AF)/[(BW)(1000)]$$

Where:

D_t = Dose via ingestion of poultry, mg/(kg-d)

C_t = Contaminant concentration in poultry, µg/kg

I_t = Ingestion rate, poultry, kg/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

It was assumed that the total daily intake of poultry, (I_t) is 0.069 kg/d (Statistics Canada 1986). This value represents the average rate of consumption of poultry for the total Canadian population.

I.2.7 INGESTION OF EGGS

Long-term exposures through ingestion of eggs taken from chickens raised in the vicinity of the proposed facility were calculated using the following equation:

$$D_e = (C_e)(I_e)(AF)/[(BW)(1000)]$$

Where:

D_e = Dose via ingestion of eggs, mg/(kg-d)

C_e = Contaminant concentration in poultry, µg/kg

I_e = Ingestion rate, eggs, kg/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

It was assumed in the risk assessment that the total daily intake of eggs, (I_e) is 0.033 kg/d (Statistics Canada 1986). This value represents the average rate of consumption of eggs for the total Canadian population.

I.2.8 INGESTION OF MILK

Long-term exposures through ingestion of milk and milk products taken from dairy cows raised in the vicinity of the proposed facility were calculated using the following equation:

$$D_m = (C_m)(I_m)(AF)/[(BW)(1000)]$$

Where:

D_m = Dose via ingestion of milk products, mg/(kg-d)

C_m = Contaminant concentration in milk, μg/kg

I_m = Ingestion rate, milk products, kg/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

It was assumed in the risk assessment that the total daily intake of milk, cheese, and other milk products is 0.381 kg/d (Statistics Canada 1986). This is the average rate of consumption of milk and milk products for the total Canadian population.

I.2.9 INGESTION OF FRUITS AND VEGETABLES

Long-term exposures through ingestion of fruits and vegetables raised in the vicinity of the proposed facility were calculated using the following equation:

$$Dfv = [(C_{ep})(I_{ep})+(C_{up})(I_{up})+(C_{l})(I_{l})](AF)/[(BW)(1000)]$$

Where:

D_{fv} = Dose via ingestion of fruits and vegetables, mg/(kg-d)

C_{ep} = Contaminant concentration in exposed produce, µg/kg

I_{ep} = Ingestion rate, exposed produce, kg/d

C_{up} = Contaminant concentration in unexposed produce, µg/kg

I_{up} = Ingestion rate, unexposed produce, kg/d

C_{l} = Contaminant concentration in leafy vegetables, µg/kg

I_{l} = Ingestion rate, leafy vegetables, kg/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

As shown in the equation, three types of produce were considered in the risk assessment:

- Leafy vegetables, which present a leafy, edible surface exposed to the atmosphere. Examples include lettuce and broccoli.
- Exposed produce, which also presents edible surfaces exposed to the atmosphere but which is not leafy in nature. Examples include tomatoes and apples.
- Unexposed produce, which does not have edible surfaces exposed to the atmosphere. Examples include potatoes and corn.

These types of produce were considered separately in the risk assessment because of differences in the methods used to predict chemical concentrations in the edible portions (see Appendix D).

The following assumptions were made in calculating the dose via ingestion of fruit and vegetables:

- I_{ep} = 0.271 kg/d. The total daily intake of exposed produce (Derived from Statistics Canada 1987).
- I_{up} = 0.271 kg/d. The total daily intake of protected produce (Derived from Statistics Canada 1987).
- I_{l} = 0.071 kg/d. The total daily intake of leafy vegetables (Derived from Statistics Canada 1987).

These values correspond to average consumption rates of these three types of produce for the total Canadian population.

I.2.10 INGESTION OF POND AND CISTERN WATER

Long-term exposures through ingestion of pond and cistern water in the vicinity of the proposed facility were calculated using the following equations:

$$D_{pw} = (C_{pw})(I_w)(AF)/[(BW)(1000)]$$

$$D_{cw} = (C_{cw})(I_w)(AF)/[(BW)(1000)]$$

Where:

D_{pw} = Dose via ingestion of pond water, mg/(kg-d)

D_{cw} = Dose via ingestion of cistern water, mg/(kg-d)

C_{pw} = Contaminant concentration in pond water, µg/l

C_{cw} = Contaminant concentration in cistern water, µg/l

I_w = Ingestion rate, water, l/d

AF = Absorption factor, dimensionless

BW = Body weight, kg

It was assumed in the risk assessment that the total daily intake of drinking water (I_w) is 2 l/d (USEPA, 1980).

I.2.11 INGESTION OF SOIL

Long-term exposures through the incidental ingestion of soil in the vicinity of the proposed facility were calculated separately for children and for adults using the following equations:

$$D_{sc} = (C_s)(I_{sc})(AF)/[(BW_c)(1x10^{+9})]$$

$$D_{sa} = (C_s)(I_{sa})(AF)/[(BW_a)(1x10^{+9})]$$

Where:

D_{sc} = Dose via ingestion of soil for a child, mg/(kg-d)

D_{sa} = Dose via ingestion of soil for an adult, mg/(kg-d)

C_s = Contaminant concentration in soil, µg/kg

I_{sc} = Ingestion rate of soil for a child, mg/d

I_{sa} = Ingestion rate of soil for an adult, mg/d

AF = Absorption factor, dimensionless

BW_c = Body weight of a child, kg

BW_a = Body weight of an adult, kg

The total lifetime average dose was then calculated using the following equation:

$$D_s = (D_{sc})(F_c)+(D_{sa})(F_a)$$

Where:

D_{sc} = Dose via ingestion of soil for a child, mg/(kg-d)

D_{sa} = Dose via ingestion of soil for an adult, mg/(kg-d)

F_c = Fraction of a 70 year lifespan that the person is considered a child, unitless

F_a = Fraction of a 70 year lifespan that the person is considered an adult, unitless

The following assumptions were made in calculating the dose via ingestion of soil:

- I_{sc} = 100 mg/d. The total daily intake of soil by a typical child (USEPA 1984).
- I_{sa} = 20 mg/d. The total daily intake of soil by a typical adult (USEPA 1984).
- F_c = (10/70) = 0.14
- F_a = (60/70) = 0.86
- BW_c = 31 kg. A body weight of 31 kg, which approximates the average weight of a child over the first 9 years of life, was assumed (ICRP 1984).
- BW_a = 70 kg. A body weight of 70 kg, which approximates the average weight of a man over the course of a lifetime, was assumed (NIOSH 1980).

I.2.12 DERMAL CONTACT WITH SOIL

Long-term exposures through dermal contact with soil in the vicinity of the proposed facility were calculated separately for children and for adults using the following equations:

$$D_{dc} = (C_s)(DP_c)(EX_c)(AF)/[(BW_c)(1x10^{+9})]$$

$$D_{da} = (C_s)(DP_a)(EX_a)(AF)/[(BW_a)(1x10^{+9})]$$

Where:

C_s = Contaminant concentration in soil, µg/kg

DP_c = Soil deposition onto skin for a child, mg/(cm^2-d)

DP_a = Soil deposition onto skin for an adult, mg/(cm^2-d)

EX_c = Skin area exposed for a child, cm^2

EX_a = Skin area exposed for an adult, cm^2

BW_c = Body weight of a child, kg

BW_a = Body weight of an adult, kg

AF = Absorption factor for dermal contact

The total lifetime average dose was then calculated using the following equation:

$$D_s = (D_{dc})(F_c)+(D_{da})(F_a)$$

Where:

D_{dc} = Dose via dermal contact with soil for a child, mg/(kg-d)

D_{da} = Dose via dermal contact with soil for an adult, mg/(kg-d)

F_c = Fraction of a 70 year lifespan that the person is considered a child, unitless

F_a = Fraction of a 70 year lifespan that the person is considered an adult, unitless

The following assumptions were made in calculating the dose via dermal contact with soil:

- AF = 0.10
- DP_c = 0.5 mg/(cm^2-d). (Lepow et al. 1975)
- DP_a = 0.5 mg/(cm^2-d). (Lepow et al 1975)
- EX_c = 3105 cm^2. A skin area of 3105 cm^2 corresponds to the face, two-thirds of the upper limbs and one-half of the lower limbs of a nine year old child. (ICRP 1984)
- EX_a = 2970 cm^2. A skin area of 2970 cm^2 corresponds to the face and two-thirds of the upper limbs of an adult male. (ICRP 1984)
- F_c = (10/70) = 0.14
- F_a = (60/70) = 0.86
- BW_c = 31 kg. A body weight of 31 kg, which approximates the average weight of a child over the first 9 years of life, was assumed (ICRP 1984).
- BW_a = 70 kg. A body weight of 70 kg, which approximates the average weight of a man over the course of a lifetime, was assumed (NIOSH 1980).

I.2.13 SUMMARY OF HUMAN DOSES ASSOCIATED WITH ROUTINE ATMOSPHERIC RELEASES

The predicted human doses associated with each exposure pathway are presented in Table I-1 through I-18. Doses for each individual pathway are presented for each emission source. The chemical concentrations in the various environmental media that were used as input to the calculation of human doses are presented in Appendix D.

TABLE I-1

HUMAN EXPOSURE TO INCINERATOR EMISSIONS VIA INHALATION

CHEMICAL CONSTITUENT	ANNUAL CHRONIC DOSE (mg/kg/d)	MAX 2 WEEK CHRONIC DOSE (mg/kg/d)
ACETONE	2.60E-07	2.49E-06
ACRYLONITRILE	1.62E-09	1.55E-08
BENZENE	5.44E-08	5.20E-07
BENZO(A)ANTHRACENE	1.23E-10	1.17E-09
BENZO(A)PYRENE	4.90E-11	4.68E-10
BROMODICHLOROMETHANE	1.85E-09	1.77E-08
BROMOFORM	1.43E-10	1.37E-09
BUTADIENE,1,3-	1.45E-10	1.38E-09
CARBON TETRACHLORIDE	8.21E-09	7.85E-08
CHLOROBENZENE	5.88E-10	5.62E-09
CHLOROFORM	5.96E-08	5.70E-07
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	6.55E-14	6.27E-13
DIOXIN,2,3,7,8-EQ(MOE)	1.35E-12	1.29E-11
ETHANOL	3.82E-07	3.66E-06
ETHYLENE GLYCOL	4.81E-07	4.60E-06
ETHYLENE OXIDE	1.24E-10	1.18E-09
FORMALDEHYDE	4.37E-09	4.18E-08
HEXACHLOROBENZENE	6.97E-11	6.66E-10
HYDRAZINE	4.04E-13	3.86E-12
METHYL ETHYL KETONE	7.06E-08	6.75E-07
METHYLENE CHLORIDE	1.37E-07	1.31E-06
NAPHTHALENE	5.63E-09	5.38E-08
NITROPHENOL,o-	4.03E-09	3.86E-08
PCB	1.75E-10	1.67E-09
PETROLEUM DISTILLATES	4.21E-07	4.03E-06
PHENOL	1.14E-09	1.09E-08
PYRIDINE	8.11E-09	7.75E-08
STYRENE	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	1.16E-07	1.11E-06
TETRAETHYL LEAD	1.29E-11	1.24E-10
TOLUENE	3.67E-07	3.51E-06
TRICHLOROETHANE,1,1,1-	1.42E-07	1.36E-06
TRICHLOROETHYLENE	1.27E-07	1.21E-06
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00
XYLENE	1.85E-07	1.77E-06
ARSENIC	5.36E-10	5.12E-09
BERYLLIUM	4.28E-12	4.10E-11
CADMIUM	9.49E-09	9.08E-08
CHROMIUM (VI)	2.25E-08	2.15E-07
COPPER	5.29E-08	5.06E-07
LEAD	1.47E-07	1.40E-06
MERCURY	8.74E-09	8.36E-08
NICKEL	6.39E-09	6.11E-08
ZINC	0.00E+00	0.00E+00

TABLE I-2

HUMAN EXPOSURE TO INCINERATOR EMISSIONS VIA INGESTION OF PRODUCE, MEAT, AND ANIMAL PRODUCTS

CHEMICAL CONSTITUENT	PRODUCE DOSE (mg/kg-d)	BEEF DOSE (mg/kg-d)	MILK DOSE (mg/kg-d)	PORK DOSE (mg/kg-d)	POULTRY DOSE (mg/kg-d)	EGG DOSE (mg/kg-d)
ACETONE	1.76E-06	5.97E-13	2.92E-12	1.58E-10	1.22E-10	6.65E-11
ACRYLONITRILE	4.85E-09	5.16E-15	2.50E-14	8.76E-13	6.27E-13	3.42E-13
BENZENE	3.57E-09	4.00E-13	1.56E-12	9.70E-12	7.71E-12	4.20E-12
BENZO(A)ANTHRACENE	2.20E-09	2.41E-09	4.05E-09	1.49E-09	3.09E-09	1.68E-09
BENZO(A)PYRENE	9.11E-10	2.81E-09	4.79E-09	1.15E-09	2.17E-09	1.18E-09
BROMODICHLOROMETHANE	5.20E-10	5.01E-14	2.07E-13	1.33E-12	8.61E-13	4.69E-13
BROMOFORM	1.22E-10	2.32E-14	9.45E-14	4.66E-13	2.49E-13	1.36E-13
BUTADIENE,1,3-	3.83E-12	4.65E-16	1.39E-15	9.36E-15	1.75E-14	9.54E-15
CARBON TETRACHLORIDE	5.68E-10	4.34E-13	1.42E-12	4.31E-12	4.01E-12	2.19E-12
CHLOROBENZENE	2.17E-10	1.45E-13	5.26E-13	1.61E-12	8.76E-13	4.78E-13
CHLOROFORM	1.50E-08	1.09E-12	4.43E-12	3.23E-11	2.39E-11	1.31E-11
DICHLORODIFLUOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	8.63E-14	3.85E-12	1.28E-12	2.93E-13	5.12E-13	2.79E-13
DIOXIN,2,3,7,8-EQ(MOE)	1.79E-12	1.31E-11	5.33E-12	6.09E-12	1.06E-11	5.80E-12
ETHANOL	2.91E-06	8.20E-13	1.00E-12	2.32E-10	1.84E-10	1.00E-10
ETHYLENE GLYCOL	3.10E-05	3.79E-13	2.83E-13	3.01E-10	1.20E-09	6.56E-10
ETHYLENE OXIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
FORMALDEHYDE	2.66E-08	3.49E-15	4.29E-15	1.32E-12	1.11E-12	6.03E-13
HEXACHLOROBENZENE	7.97E-09	6.41E-08	2.11E-08	9.90E-09	3.53E-09	1.92E-09
HYDRAZINE	1.01E-11	2.52E-19	3.11E-19	1.81E-16	1.71E-16	9.33E-17
METHYL ETHYL KETONE	2.67E-07	2.90E-13	3.53E-13	4.88E-11	3.48E-11	1.89E-11
METHYLENE CHLORIDE	2.88E-08	3.64E-13	4.29E-13	2.23E-11	1.78E-11	9.73E-12
NAPHTHALENE	6.02E-09	1.42E-11	1.21E-11	8.52E-11	4.09E-11	2.23E-11
NITROPHENOL,o-	7.04E-09	2.70E-13	3.04E-13	1.09E-11	6.35E-12	3.46E-12
PCB	6.18E-10	1.07E-08	1.37E-09	6.34E-10	8.42E-10	4.59E-10
PETROLEUM DISTILLATES	9.44E-08	2.94E-13	3.54E-13	3.14E-11	2.75E-11	1.50E-11
PHENOL	2.87E-09	4.39E-11	7.66E-11	1.14E-09	3.12E-09	1.70E-09
PYRIDINE	2.61E-08	6.94E-14	8.33E-14	8.23E-12	5.63E-12	3.07E-12
STYRENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TETRACHLOROETHYLENE	2.68E-08	1.01E-11	1.03E-11	1.43E-10	1.05E-10	5.74E-11
TETRAETHYL LEAD	1.04E-11	1.42E-12	8.49E-13	1.03E-12	7.89E-13	4.30E-13
TOLUENE	8.04E-08	3.67E-11	3.67E-11	4.03E-10	3.00E-10	1.63E-10
TRICHLOROETHANE,1,1,1-	9.86E-09	3.25E-12	3.41E-12	4.72E-11	4.65E-11	2.54E-11
TRICHLOROETHYLENE	1.87E-08	4.54E-12	4.81E-12	7.75E-11	6.44E-11	3.51E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
XYLENE	3.23E-08	4.40E-11	4.04E-11	3.42E-10	2.13E-10	1.16E-10
ARSENIC	1.43E-08	3.67E-07	5.93E-08	5.39E-08	1.20E-09	5.69E-10
BERYLLIUM	1.32E-10	1.65E-11	7.29E-14	1.94E-12	7.52E-14	3.57E-14
CADMIUM	8.94E-08	5.26E-09	5.67E-08	8.28E-10	1.94E-11	9.22E-12
CHROMIUM (VI)	2.90E-07	1.85E-06	2.70E-06	2.67E-07	6.26E-09	2.97E-09
COPPER	2.04E-06	3.14E-06	2.57E-06	4.71E-07	1.15E-08	5.48E-09
LEAD	8.61E-06	4.23E-07	1.87E-06	5.64E-08	1.70E-09	8.05E-10
MERCURY	6.21E-07	1.02E-05	1.05E-07	1.67E-06	5.31E-08	2.52E-08
NICKEL	3.64E-07	2.66E-06	2.39E-06	3.90E-07	8.85E-09	4.20E-09
ZINC	2.57E-04	5.91E-04	3.94E-04	1.08E-04	2.64E-06	1.25E-06

TABLE I-3

HUMAN EXPOSURE TO INCINERATOR EMISSIONS VIA INGESTION OF SOIL

CHEMICAL CONSTITUENT	SOIL INGESTION DOSE (mg/kg-d)
ACETONE	2.98E-10
ACRYLONITRILE	1.62E-12
BENZENE	2.47E-11
BENZO(A)ANTHRACENE	2.36E-11
BENZO(A)PYRENE	1.13E-11
BROMODICHLOROMETHANE	1.73E-12
BROMOFORM	5.82E-13
BUTADIENE,1,3-	1.11E-14
CARBON TETRACHLORIDE	4.79E-12
CHLOROBENZENE	1.86E-12
CHLOROFORM	4.25E-11
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.41E-15
DIOXIN,2,3,7,8-EQ(MOE)	2.92E-14
ETHANOL	4.45E-10
ETHYLENE GLYCOL	4.71E-10
ETHYLENE OXIDE	1.44E-13
FORMALDEHYDE	2.67E-12
HEXACHLOROBENZENE	1.75E-11
HYDRAZINE	4.08E-16
METHYL ETHYL KETONE	8.55E-11
METHYLENE CHLORIDE	3.29E-11
NAPHTHALENE	9.10E-11
NITROPHENOL,o-	1.50E-11
PCB	2.19E-11
PETROLEUM DISTILLATES	5.09E-11
PHENOL	4.15E-12
PYRIDINE	1.35E-11
STYRENE	0.00E+00
TETRACHLOROETHYLENE	1.69E-10
TETRAETHYL LEAD	7.46E-13
TOLUENE	5.68E-10
TRICHLOROETHANE,1,1,1-	5.53E-11
TRICHLOROETHYLENE	9.37E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	3.75E-10
ARSENIC	1.87E-10
BERYLLIUM	2.32E-12
CADMIUM	2.19E-09
CHROMIUM (VI)	9.54E-09
COPPER	8.31E-09
LEAD	1.19E-07
MERCURY	7.02E-10
NICKEL	2.06E-09
ZINC	1.39E-07

TABLE I-4

HUMAN EXPOSURE TO INCINERATOR EMISSIONS VIA DERMAL CONTACT WITH SOIL

CHEMICAL CONSTITUENT	SOIL CONTACT DOSE (mg/kg-d)
ACETONE	1.07E-09
ACRYLONITRILE	5.80E-12
BENZENE	8.89E-11
BENZO(A)ANTHRACENE	8.47E-11
BENZO(A)PYRENE	4.04E-11
BROMODICHLOROMETHANE	6.22E-12
BROMOFORM	2.09E-12
BUTADIENE,1,3-	3.99E-14
CARBON TETRACHLORIDE	1.72E-11
CHLOROBENZENE	6.66E-12
CHLOROFORM	1.53E-10
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	2.02E-14
DIOXIN,2,3,7,8-EQ(MOE)	4.20E-13
ETHANOL	1.60E-09
ETHYLENE GLYCOL	1.69E-09
ETHYLENE OXIDE	5.18E-13
FORMALDEHYDE	9.59E-12
HEXACHLOROBENZENE	6.28E-11
HYDRAZINE	1.46E-15
METHYL ETHYL KETONE	3.07E-10
METHYLENE CHLORIDE	1.18E-10
NAPHTHALENE	3.27E-10
NITROPHENOL,o-	5.38E-11
PCB	7.85E-11
PETROLEUM DISTILLATES	1.83E-10
PHENOL	1.49E-11
PYRIDINE	4.86E-11
STYRENE	0.00E+00
TETRACHLOROETHYLENE	6.07E-10
TETRAETHYL LEAD	2.68E-12
TOLUENE	2.04E-09
TRICHLOROETHANE,1,1,1-	1.99E-10
TRICHLOROETHYLENE	3.36E-10
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	1.35E-09
ARSENIC	6.72E-10
BERYLLIUM	8.34E-12
CADMIUM	7.86E-09
CHROMIUM (VI)	3.42E-08
COPPER	2.98E-08
LEAD	4.29E-07
MERCURY	2.52E-09
NICKEL	7.40E-09
ZINC	5.00E-07

TABLE I-5

HUMAN EXPOSURE TO INCINERATOR EMISSIONS VIA INGESTION OF POND WATER

CONTAMINANT	POND DOSE (mg/kg-d)
ACETONE	1.86E-07
ACRYLONITRILE	1.90E-10
BENZENE	1.29E-09
BENZO(A)ANTHRACENE	9.47E-10
BENZO(A)PYRENE	1.33E-09
BROMODICHLOROMETHANE	9.81E-11
BROMOFORM	7.58E-12
BUTADIENE,1,3-	6.71E-12
CARBON TETRACHLORIDE	3.89E-10
CHLOROBENZENE	2.79E-11
CHLOROFORM	4.04E-09
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	3.06E-14
DIOXIN,2,3,7,8-EQ(MOE)	6.35E-13
ETHANOL	2.74E-07
ETHYLENE GLYCOL	2.24E-08
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	5.39E-10
HEXACHLOROBENZENE	6.27E-12
HYDRAZINE	1.88E-14
METHYL ETHYL KETONE	8.70E-09
METHYLENE CHLORIDE	7.26E-09
NAPHTHALENE	2.99E-10
NITROPHENOL,o-	2.89E-09
PCB	6.69E-12
PETROLEUM DISTILLATES	1.97E-08
PHENOL	9.90E-09
PYRIDINE	3.77E-10
STYRENE	0.00E+00
TETRACHLOROETHYLENE	9.69E-09
TETRAETHYL LEAD	1.51E-12
TOLUENE	1.74E-08
TRICHLOROETHANE,1,1,1-	6.74E-09
TRICHLOROETHYLENE	8.61E-09
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	8.77E-09
ARSENIC	7.16E-09
BERYLLIUM	7.67E-11
CADMIUM	1.26E-08
CHROMIUM (VI)	1.55E-07
COPPER	2.89E-07
LEAD	3.88E-06
MERCURY	1.94E-07
NICKEL	8.46E-08
ZINC	1.09E-05

TABLE I-6

HUMAN EXPOSURE TO INCINERATOR EMISSIONS VIA INGESTION OF CISTERN WATER

CONTAMINANT	CISTERN DOSE (mg/kg-d)
ACETONE	1.81E-08
ACRYLONITRILE	3.72E-11
BENZENE	1.05E-11
BENZO(A)ANTHRACENE	2.56E-10
BENZO(A)PYRENE	2.09E-10
BROMODICHLOROMETHANE	1.61E-12
BROMOFORM	5.33E-13
BUTADIENE,1,3-	1.48E-15
CARBON TETRACHLORIDE	5.74E-13
CHLOROBENZENE	3.49E-13
CHLOROFORM	4.94E-11
DICHLORODIFLUOROMETHANE	0.00E+00
DIOXIN,2,3,7,8-EQ(EPA)	1.90E-13
DIOXIN,2,3,7,8-EQ(MOE)	3.94E-12
ETHANOL	2.28E-07
ETHYLENE GLYCOL	7.29E-03
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	3.30E-11
HEXACHLOROBENZENE	2.18E-13
HYDRAZINE	6.51E-11
METHYL ETHYL KETONE	4.09E-09
METHYLENE CHLORIDE	1.33E-10
NAPHTHALENE	2.60E-11
NITROPHENOL,o-	1.11E-09
PCB	1.40E-13
PETROLEUM DISTILLATES	1.54E-13
PHENOL	6.68E-09
PYRIDINE	2.43E-06
STYRENE	0.00E+00
TETRACHLOROETHYLENE	2.46E-11
TETRAETHYL LEAD	8.58E-16
TOLUENE	1.15E-10
TRICHLOROETHANE,1,1,1-	1.76E-11
TRICHLOROETHYLENE	4.03E-11
TRICHLOROTRIFLUOROETHANE	0.00E+00
XYLENE	7.31E-11
ARSENIC	7.67E-08
BERYLLIUM	8.15E-10
CADMIUM	1.35E-06
CHROMIUM (VI)	3.29E-06
COPPER	7.74E-06
LEAD	4.11E-05
MERCURY	2.08E-06
NICKEL	9.06E-07
ZINC	1.17E-04

TABLE I-7

HUMAN EXPOSURE TO EVAPORATOR EMISSIONS VIA INHALATION

CHEMICAL CONSTITUENT	ANNUAL CHRONIC DOSE (mg/kg/d)	MAX 2 WEEK CHRONIC DOSE (mg/kg/d)
ACETONE	0.00E+00	0.00E+00
ACRYLONITRILE	2.16E-08	1.01E-07
BENZENE	4.17E-09	1.96E-08
BENZO(A)ANTHRACENE	4.02E-12	1.88E-11
BENZO(A)PYRENE	1.08E-11	5.07E-11
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	1.05E-09	4.93E-09
CARBON TETRACHLORIDE	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	2.78E-07	1.30E-06
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00
FORMALDEHYDE	1.00E-10	4.71E-10
HEXACHLOROBENZENE	0.00E+00	0.00E+00
HYDRAZINE	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00
NAPHTHALENE	2.78E-07	1.30E-06
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	4.17E-12	1.96E-11
PETROLEUM DISTILLATES	0.00E+00	0.00E+00
PHENOL	2.47E-09	1.16E-08
PYRIDINE	0.00E+00	0.00E+00
STYRENE	2.65E-09	1.24E-08
TETRACHLOROETHYLENE	8.01E-09	3.76E-08
TETRAETHYL LEAD	0.00E+00	0.00E+00
TOLUENE	7.41E-08	3.48E-07
TRICHLOROETHANE,1,1,1-	2.78E-07	1.30E-06
TRICHLOROETHYLENE	4.97E-09	2.33E-08
TRICHLOROTRIFLUOROETHANE	2.47E-07	1.16E-06
XYLENE	1.41E-08	6.60E-08
ARSENIC	4.33E-13	2.03E-12
BERYLLIUM	0.00E+00	0.00E+00
CADMIUM	1.02E-14	4.78E-14
CHROMIUM (VI)	5.10E-13	2.39E-12
COPPER	0.00E+00	0.00E+00
LEAD	0.00E+00	0.00E+00
MERCURY	0.00E+00	0.00E+00
NICKEL	5.10E-13	2.39E-12
ZINC	0.00E+00	0.00E+00

TABLE I-8

HUMAN EXPOSURE TO EVAPORATOR EMISSIONS VIA INGESTION OF PRODUCE, MEAT, AND ANIMAL PRODUCTS

CHEMICAL CONSTITUENT	PRODUCE DOSE (mg/kg-d)	BEEF DOSE (mg/kg-d)	MILK DOSE (mg/kg-d)	PORK DOSE (mg/kg-d)	POULTRY DOSE (mg/kg-d)	EGG DOSE (mg/kg-d)
ACETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ACRYLONITRILE	6.81E-08	7.24E-14	3.50E-13	1.23E-11	8.79E-12	4.79E-12
BENZENE	5.48E-10	6.10E-14	2.39E-13	1.48E-12	1.17E-12	6.38E-13
BENZO(A)ANTHRACENE	7.19E-11	7.82E-11	1.31E-10	4.83E-11	1.01E-10	5.48E-11
BENZO(A)PYRENE	2.10E-10	6.43E-10	1.10E-09	2.63E-10	4.97E-10	2.71E-10
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.79E-11	3.37E-15	1.01E-14	6.79E-14	1.27E-13	6.91E-14
CARBON TETRACHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DICHLORODIFLUOROMETHANE	4.86E-10	5.24E-13	7.41E-13	3.52E-12	2.90E-11	1.58E-11
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE GLYCOL	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHYLENE OXIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
FORMALDEHYDE	6.10E-10	8.00E-17	3.92E-16	3.04E-14	2.53E-14	1.38E-14
HEXACHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYL ETHYL KETONE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
METHYLENE CHLORIDE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
NAPHTHALENE	2.97E-07	6.91E-10	2.27E-09	4.15E-09	1.90E-09	1.03E-09
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	1.48E-11	2.48E-10	1.16E-10	1.45E-11	1.93E-11	1.05E-11
PETROLEUM DISTILLATES	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PHENOL	6.21E-09	4.40E-11	7.71E-11	1.14E-09	3.12E-09	1.70E-09
PYRIDINE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
STYRENE	9.88E-10	9.01E-13	3.17E-12	8.53E-12	4.85E-12	2.64E-12
TETRACHLOROETHYLENE	1.85E-09	6.88E-13	2.51E-12	9.78E-12	7.11E-12	3.87E-12
TETRAETHYL LEAD	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
TOLUENE	1.63E-08	7.34E-12	2.72E-11	9.69E-11	5.90E-11	3.22E-11
TRICHLOROETHANE,1,1,1-	1.93E-08	6.37E-12	2.19E-11	9.26E-11	9.15E-11	4.99E-11
TRICHLOROETHYLENE	7.32E-10	1.76E-13	6.48E-13	3.02E-12	2.49E-12	1.36E-12
TRICHLOROTRIFLUOROETHAN	9.11E-10	1.27E-11	2.06E-11	3.00E-11	1.52E-10	8.27E-11
XYLENE	2.46E-09	3.30E-12	1.11E-11	2.57E-11	1.56E-11	8.51E-12
ARSENIC	1.15E-11	3.63E-07	5.88E-08	5.35E-08	1.18E-09	5.62E-10
BERYLLIUM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CADMIUM	9.59E-13	3.33E-08	3.27E-07	4.90E-09	1.08E-10	5.15E-11
CHROMIUM (VI)	1.31E-11	3.33E-06	4.90E-06	4.90E-07	1.08E-08	5.15E-09
COPPER	0.00E+00	6.06E-06	4.90E-06	8.91E-07	1.97E-08	9.36E-09
LEAD	0.00E+00	1.64E-07	7.35E-07	2.41E-08	5.32E-10	2.53E-10
MERCURY	0.00E+00	6.06E-07	5.88E-09	8.91E-08	1.97E-09	9.36E-10
NICKEL	2.91E-11	2.54E-06	2.29E-06	3.74E-07	8.28E-09	3.93E-09
ZINC	0.00E+00	1.03E-04	5.55E-05	1.52E-05	3.35E-07	1.59E-07

TABLE I-9

HUMAN EXPOSURE TO EVAPORATOR EMISSIONS VIA INGESTION OF SOIL

CHEMICAL CONSTITUENT	SOIL INGESTION DOSE (mg/kg-d)
ACETONE	0.00E+00
ACRYLONITRILE	1.83E-11
BENZENE	1.61E-12
BENZO(A)ANTHRACENE	6.54E-13
BENZO(A)PYRENE	2.20E-12
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	6.85E-14
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	1.34E-12
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	5.20E-14
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	3.81E-09
NITROPHENOL,o-	0.00E+00
PCB	4.42E-13
PETROLEUM DISTILLATES	0.00E+00
PHENOL	7.62E-12
PYRIDINE	0.00E+00
STYRENE	8.24E-12
TETRACHLOROETHYLENE	9.86E-12
TETRAETHYL LEAD	0.00E+00
TOLUENE	9.75E-11
TRICHLOROETHANE,1,1,1-	9.18E-11
TRICHLOROETHYLENE	3.11E-12
TRICHLOROTRIFLUOROETHAN	1.33E-11
XYLENE	2.42E-11
ARSENIC	1.28E-13
BERYLLIUM	0.00E+00
CADMIUM	1.99E-15
CHROMIUM (VI)	1.83E-13
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	1.40E-13
ZINC	0.00E+00

TABLE I-10

HUMAN EXPOSURE TO EVAPORATOR EMISSIONS VIA DERMAL CONTACT WITH SOIL

CHEMICAL CONSTITUENT	SOIL CONTACT DOSE (mg/kg-d)
ACETONE	0.00E+00
ACRYLONITRILE	6.23E-11
BENZENE	5.49E-12
BENZO(A)ANTHRACENE	2.23E-12
BENZO(A)PYRENE	7.49E-12
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	2.34E-13
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	4.56E-12
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	1.77E-13
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	1.30E-08
NITROPHENOL,o-	0.00E+00
PCB	1.51E-12
PETROLEUM DISTILLATES	0.00E+00
PHENOL	2.60E-11
PYRIDINE	0.00E+00
STYRENE	2.81E-11
TETRACHLOROETHYLENE	3.36E-11
TETRAETHYL LEAD	0.00E+00
TOLUENE	3.32E-10
TRICHLOROETHANE,1,1,1-	3.13E-10
TRICHLOROETHYLENE	1.06E-11
TRICHLOROTRIFLUOROETHAN	4.54E-11
XYLENE	8.24E-11
ARSENIC	4.38E-13
BERYLLIUM	0.00E+00
CADMIUM	6.79E-15
CHROMIUM (VI)	6.24E-13
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	4.76E-13
ZINC	0.00E+00

TABLE I-11

HUMAN EXPOSURE TO EVAPORATOR EMISSIONS VIA INGESTION OF POND WATER

CONTAMINANT	POND DOSE (mg/kg-d)
ACETONE	0.00E+00
ACRYLONITRILE	2.67E-09
BENZENE	1.98E-10
BENZO(A)ANTHRACENE	3.09E-11
BENZO(A)PYRENE	3.06E-10
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	4.88E-11
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	1.29E-08
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	1.24E-11
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	1.48E-08
NITROPHENOL,o-	0.00E+00
PCB	1.60E-13
PETROLEUM DISTILLATES	0.00E+00
PHENOL	2.14E-08
PYRIDINE	0.00E+00
STYRENE	1.90E-10
TETRACHLOROETHYLENE	6.67E-10
TETRAETHYL LEAD	0.00E+00
TOLUENE	3.52E-09
TRICHLOROETHANE,1,1,1-	1.32E-08
TRICHLOROETHYLENE	3.37E-10
TRICHLOROTRIFLUOROETHAN	1.29E-08
XYLENE	6.67E-10
ARSENIC	5.79E-12
BERYLLIUM	0.00E+00
CADMIUM	1.35E-13
CHROMIUM (VI)	7.03E-12
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	6.75E-12
ZINC	0.00E+00

TABLE I-12

HUMAN EXPOSURE TO EVAPORATOR EMISSIONS VIA INGESTION OF CISTERN WATER

CONTAMINANT	CISTERN DOSE (mg/kg-day)
ACETONE	0.00E+00
ACRYLONITRILE	5.22E-10
BENZENE	1.61E-12
BENZO(A)ANTHRACENE	8.37E-12
BENZO(A)PYRENE	4.80E-11
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.08E-14
CARBON TETRACHLORIDE	0.00E+00
CHLOROBENZENE	0.00E+00
CHLOROFORM	0.00E+00
DICHLORODIFLUOROMETHANE	2.77E-13
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	0.00E+00
ETHYLENE GLYCOL	0.00E+00
ETHYLENE OXIDE	0.00E+00
FORMALDEHYDE	7.58E-13
HEXACHLOROBENZENE	0.00E+00
METHYL ETHYL KETONE	0.00E+00
METHYLENE CHLORIDE	0.00E+00
NAPHTHALENE	1.29E-09
NITROPHENOL,o-	0.00E+00
PCB	3.35E-15
PETROLEUM DISTILLATES	0.00E+00
PHENOL	1.44E-08
PYRIDINE	0.00E+00
STYRENE	1.86E-12
TETRACHLOROETHYLENE	1.69E-12
TETRAETHYL LEAD	0.00E+00
TOLUENE	2.32E-11
TRICHLOROETHANE,1,1,1-	3.44E-11
TRICHLOROETHYLENE	1.58E-12
TRICHLOROTRIFLUOROETHAN	1.98E-12
XYLENE	5.55E-12
ARSENIC	6.20E-11
BERYLLIUM	0.00E+00
CADMIUM	1.44E-12
CHROMIUM (VI)	7.45E-11
COPPER	0.00E+00
LEAD	0.00E+00
MERCURY	0.00E+00
NICKEL	7.22E-11
ZINC	0.00E+00

TABLE I-13

HUMAN EXPOSURE TO FUGITIVE EMISSIONS VIA INHALATION

CHEMICAL CONSTITUENT	ANNUAL DOSE (mg/kg/d)	MAX 2 WEEK DOSE (mg/kg/d)
ACETONE	1.40E-05	1.06E-04
ACRYLONITRILE	1.45E-07	1.16E-06
BENZENE	5.68E-07	4.30E-06
BENZO(A)ANTHRACENE	6.40E-19	4.88E-18
BENZO(A)PYRENE	2.55E-19	1.95E-18
BROMODICHLOROMETHANE	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00
BUTADIENE,1,3-	2.21E-07	1.82E-06
CARBON TETRACHLORIDE	2.62E-07	1.98E-06
CHLOROBENZENE	0.00E+00	0.00E+00
CHLOROFORM	4.55E-07	3.44E-06
DICHLORODIFLUOROMETHANE	7.17E-07	5.97E-06
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00
ETHANOL	9.65E-06	7.30E-05
ETHYLENE GLYCOL	6.00E-07	4.54E-06
ETHYLENE OXIDE	2.56E-09	1.94E-08
FORMALDEHYDE	7.33E-06	5.74E-05
HEXACHLOROBENZENE	9.27E-16	7.02E-15
HYDRAZINE	7.86E-12	5.95E-11
METHYL ETHYL KETONE	2.21E-06	1.67E-05
METHYLENE CHLORIDE	1.32E-05	9.97E-05
NAPHTHALENE	1.96E-11	1.63E-10
NITROPHENOL,o-	0.00E+00	0.00E+00
PCB	1.75E-11	1.32E-10
PETROLEUM DISTILLATES	6.66E-06	5.04E-05
PHENOL	1.79E-08	1.49E-07
PYRIDINE	1.46E-07	1.11E-06
STYRENE	3.59E-09	2.99E-08
TETRACHLOROETHYLENE	2.07E-06	1.57E-05
TETRAETHYL LEAD	1.96E-10	1.49E-09
TOLUENE	9.75E-06	7.58E-05
TRICHLOROETHANE,1,1,1-	4.24E-06	3.21E-05
TRICHLOROETHYLENE	3.42E-06	2.59E-05
TRICHLOROTRIFLUOROETHANE	1.12E-07	9.34E-07
XYLENE	2.98E-06	2.26E-05

TABLE I-14

HUMAN EXPOSURE TO FUGITIVE EMISSIONS VIA INGESTION OF PRODUCE, MEAT, AND ANIMAL PRODUCTS

CHEMICAL CONSTITUENT	PRODUCE DOSE (mg/kg-d)	BEEF DOSE (mg/kg-d)	MILK DOSE (mg/kg-d)	PORK DOSE (mg/kg-d)	POULTRY DOSE (mg/kg-d)	EGG DOSE (mg/kg-d)
ACETONE	9.45E-05	3.20E-11	1.56E-10	8.46E-09	6.54E-09	3.57E-09
ACRYLONITRILE	4.56E-07	4.85E-13	2.35E-12	8.23E-11	5.89E-11	3.21E-11
BENZENE	7.46E-08	8.30E-12	3.25E-11	2.02E-10	1.59E-10	8.68E-11
BENZO(A)ANTHRACENE	1.15E-17	1.25E-17	2.10E-17	7.70E-18	1.60E-17	8.75E-18
BENZO(A)PYRENE	4.74E-18	1.45E-17	2.48E-17	5.95E-18	1.12E-17	6.13E-18
BROMODICHLOROMETHANE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BROMOFORM	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
BUTADIENE,1,3-	5.86E-09	7.10E-13	2.12E-12	1.43E-11	2.67E-11	1.45E-11
CARBON TETRACHLORIDE	1.81E-08	1.37E-11	4.50E-11	1.36E-10	1.25E-10	6.84E-11
CHLOROBENZENE	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
CHLOROFORM	1.15E-07	8.27E-12	3.37E-11	2.45E-10	1.81E-10	9.86E-11
DICHLORODIFLUOROMETHANE	1.25E-09	1.35E-12	1.91E-12	9.07E-12	7.49E-11	4.08E-11
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
ETHANOL	7.34E-05	2.07E-11	1.01E-10	5.87E-09	4.63E-09	2.52E-09
ETHYLENE GLYCOL	3.87E-05	4.72E-13	1.41E-12	3.75E-10	1.50E-09	8.18E-10
ETHYLENE OXIDE	1.91E-08	5.61E-15	2.74E-14	1.57E-12	1.22E-12	6.67E-13
FORMALDEHYDE	4.46E-05	5.85E-12	2.87E-11	2.22E-09	1.85E-09	1.01E-09
HEXACHLOROBENZENE	1.06E-13	8.50E-13	1.12E-12	1.32E-13	4.66E-14	2.54E-14
HYDRAZINE	1.97E-10	4.91E-18	2.41E-17	3.52E-15	3.33E-15	1.81E-15
METHYL ETHYL KETONE	8.34E-06	9.05E-12	4.39E-11	1.53E-09	1.08E-09	5.91E-10
METHYLENE CHLORIDE	2.77E-06	3.50E-11	1.53E-10	2.15E-09	1.71E-09	9.33E-10
NAPHTHALENE	2.10E-11	4.87E-14	1.60E-13	2.92E-13	1.34E-13	7.28E-14
NITROPHENOL,o-	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
PCB	6.19E-11	1.04E-09	4.87E-10	6.04E-11	8.07E-11	4.40E-11
PETROLEUM DISTILLATES	1.49E-06	4.65E-12	2.08E-11	4.97E-10	4.34E-10	2.37E-10
PHENOL	4.50E-08	4.48E-11	8.04E-11	1.18E-09	3.15E-09	1.72E-09
PYRIDINE	4.71E-07	1.25E-12	5.97E-12	1.48E-10	1.01E-10	5.53E-11
STYRENE	1.34E-09	1.22E-12	4.30E-12	1.16E-11	6.56E-12	3.58E-12
TETRACHLOROETHYLENE	4.76E-07	1.78E-10	6.49E-10	2.52E-09	1.83E-09	9.98E-10
TETRAETHYL LEAD	1.58E-10	2.09E-11	4.61E-11	1.46E-11	1.03E-11	5.63E-12
TOLUENE	2.14E-06	9.64E-10	3.58E-09	1.27E-08	7.75E-09	4.23E-09
TRICHLOROETHANE,1,1,1-	2.94E-07	9.61E-11	3.32E-10	1.40E-09	1.37E-09	7.47E-10
TRICHLOROETHYLENE	5.04E-07	1.21E-10	4.46E-10	2.08E-09	1.71E-09	9.32E-10
TRICHLOROTRIFLUOROETHANE	3.67E-10	5.11E-12	8.27E-12	1.20E-11	6.08E-11	3.32E-11
XYLENE	5.21E-07	6.99E-10	2.35E-09	5.44E-09	3.30E-09	1.80E-09

TABLE I-15

HUMAN EXPOSURE TO FUGITIVE EMISSIONS VIA INGESTION OF SOIL

CHEMICAL CONSTITUENT	SOIL INGESTION DOSE (mg/kg-d)
ACETONE	1.36E-08
ACRYLONITRILE	1.22E-10
BENZENE	2.19E-10
BENZO(A)ANTHRACENE	1.04E-19
BENZO(A)PYRENE	4.97E-20
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.44E-11
CARBON TETRACHLORIDE	1.30E-10
CHLOROBENZENE	0.00E+00
CHLOROFORM	2.75E-10
DICHLORODIFLUOROMETHANE	3.45E-12
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	9.53E-09
ETHYLENE GLYCOL	4.98E-10
ETHYLENE OXIDE	2.54E-12
FORMALDEHYDE	3.80E-09
HEXACHLOROBENZENE	1.98E-16
HYDRAZINE	6.73E-15
METHYL ETHYL KETONE	2.27E-09
METHYLENE CHLORIDE	2.69E-09
NAPHTHALENE	2.69E-13
NITROPHENOL,o-	0.00E+00
PCB	1.86E-12
PETROLEUM DISTILLATES	6.83E-10
PHENOL	5.52E-11
PYRIDINE	2.07E-10
STYRENE	1.12E-11
TETRACHLOROETHYLENE	2.55E-09
TETRAETHYL LEAD	9.61E-12
TOLUENE	1.28E-08
TRICHLOROETHANE,1,1,1-	1.40E-09
TRICHLOROETHYLENE	2.14E-09
TRICHLOROTRIFLUOROETHANE	5.36E-12
XYLENE	5.13E-09

TABLE I-16

HUMAN EXPOSURE TO FUGITIVE EMISSIONS VIA DERMAL CONTACT WITH SOIL

CHEMICAL CONSTITUENT	SOIL CONTACT DOSE (mg/kg-d)
ACETONE	4.63E-08
ACRYLONITRILE	4.17E-10
BENZENE	7.47E-10
BENZO(A)ANTHRACENE	3.56E-19
BENZO(A)PYRENE	1.69E-19
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	4.92E-11
CARBON TETRACHLORIDE	4.42E-10
CHLOROBENZENE	0.00E+00
CHLOROFORM	9.37E-10
DICHLORODIFLUOROMETHANE	1.18E-11
DIOXINS,2,3,7,8-EQ(EPA)	0.00E+00
DIOXINS,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	3.25E-08
ETHYLENE GLYCOL	1.70E-09
ETHYLENE OXIDE	8.65E-12
FORMALDEHYDE	1.30E-08
HEXACHLOROBENZENE	6.74E-16
HYDRAZINE	2.29E-14
METHYL ETHYL KETONE	7.73E-09
METHYLENE CHLORIDE	9.18E-09
NAPHTHALENE	9.16E-13
NITROPHENOL,o-	0.00E+00
PCB	6.33E-12
PETROLEUM DISTILLATES	2.33E-09
PHENOL	1.88E-10
PYRIDINE	7.06E-10
STYRENE	3.81E-11
TETRACHLOROETHYLENE	8.68E-09
TETRAETHYL LEAD	3.28E-11
TOLUENE	4.36E-08
TRICHLOROETHANE,1,1,1-	4.77E-09
TRICHLOROETHYLENE	7.30E-09
TRICHLOROTRIFLUOROETHANE	1.83E-11
XYLENE	1.75E-08

TABLE I-17

HUMAN EXPOSURE TO FUGITIVE EMISSIONS VIA INGESTION OF POND WATER

CONTAMINANT	POND DOSE (mg/kg-d)
ACETONE	1.00E-05
ACRYLONITRILE	1.79E-08
BENZENE	2.69E-08
BENZO(A)ANTHRACENE	4.94E-18
BENZO(A)PYRENE	6.92E-18
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	1.03E-08
CARBON TETRACHLORIDE	1.24E-08
CHLOROBENZENE	0.00E+00
CHLOROFORM	3.08E-08
DICHLORODIFLUOROMETHANE	3.33E-08
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	6.91E-06
ETHYLENE GLYCOL	2.79E-08
ETHYLENE OXIDE	3.16E-10
FORMALDEHYDE	9.04E-07
HEXACHLOROBENZENE	8.34E-17
HYDRAZINE	3.65E-13
METHYL ETHYL KETONE	2.72E-07
METHYLENE CHLORIDE	6.99E-07
NAPHTHALENE	1.04E-12
NITROPHENOL,o-	0.00E+00
PCB	6.70E-13
PETROLEUM DISTILLATES	3.12E-07
PHENOL	1.55E-07
PYRIDINE	6.79E-09
STYRENE	2.58E-10
TETRACHLOROETHYLENE	1.72E-07
TETRAETHYL LEAD	2.29E-11
TOLUENE	4.62E-07
TRICHLOROETHANE,1,1,1-	2.01E-07
TRICHLOROETHYLENE	2.32E-07
TRICHLOROTRIFLUOROETHANE	5.20E-09
XYLENE	1.41E-07

TABLE I-18

HUMAN EXPOSURE TO FUGITIVE EMISSIONS VIA INGESTION OF CISTERN WATER

CONTAMINANT	CISTERN DOSE (mg/kg-day)
ACETONE	9.72E-07
ACRYLONITRILE	3.50E-09
BENZENE	2.19E-10
BENZO(A)ANTHRACENE	1.34E-18
BENZO(A)PYRENE	1.09E-18
BROMODICHLOROMETHANE	0.00E+00
BROMOFORM	0.00E+00
BUTADIENE,1,3-	2.27E-12
CARBON TETRACHLORIDE	1.83E-11
CHLOROBENZENE	0.00E+00
CHLOROFORM	3.77E-10
DICHLORODIFLUOROMETHANE	7.13E-13
DIOXIN,2,3,7,8-EQ(EPA)	0.00E+00
DIOXIN,2,3,7,8-EQ(MOE)	0.00E+00
ETHANOL	5.75E-06
ETHYLENE GLYCOL	9.09E-03
ETHYLENE OXIDE	1.50E-10
FORMALDEHYDE	5.54E-08
HEXACHLOROBENZENE	2.90E-18
HYDRAZINE	1.27E-09
METHYL ETHYL KETONE	1.28E-07
METHYLENE CHLORIDE	1.28E-08
NAPHTHALENE	9.07E-14
NITROPHENOL,o-	0.00E+00
PCB	1.42E-14
PETROLEUM DISTILLATES	2.43E-12
PHENOL	1.05E-07
PYRIDINE	4.38E-05
STYRENE	2.53E-12
TETRACHLOROETHYLENE	4.36E-10
TETRAETHYL LEAD	1.30E-14
TOLUENE	3.05E-09
TRICHLOROETHANE,1,1,1-	5.24E-10
TRICHLOROETHYLENE	1.08E-09
TRICHLOROTRIFLUOROETHANE	7.98E-13
XYLENE	1.18E-09

I.3 SHORT-TERM EXPOSURE PATHWAYS

I.3.1 INTRODUCTION

Facility upsets, malfunctions, and accidents may lead to short-term contaminant emissions and the potential for off-site exposures. Transportation accidents involving vehicles carrying special wastes to the facility may also release potentially hazardous chemicals to the environment. The potential human health risks associated with these short-term emissions can be classified as follows:

- Acute health risks resulting from short-term exposures.
- Chronic health risks resulting from short-term exposures. For example, an exposure of limited duration to a carcinogen may contribute to the lifetime cancer risk.
- Chronic health risks resulting from long-term exposures that relate to a single release episode. For example, a spill of a persistent chemical, such as PCBs, into a stream could lead to long-term water contamination and the potential for chronic exposures.

In the risk assessment, only short-term exposures were examined in evaluating the releases that may occur as the result of upsets, malfunctions, and accidents. It was assumed that OWMC will undertake cleanup procedures to prevent long-term exposures to unacceptable levels of chemicals released during such events.

Therefore, the exposure pathways considered in the risk assessment of short-term releases were those that would exist before any mitigation activities could be implemented. For the on-site upset and accident scenarios, only direct inhalation was examined. For off-site spills, both direct inhalation and ingestion of contaminated stream water were evaluated.

I.3.2 ON-SITE UPSETS, MALFUNCTIONS, AND ACCIDENTS

The on-site upset and accident scenarios evaluated in the risk assessment for the OWMC facility are shown in Table I-19. The assumed duration of exposure associated with each event is summarized in the table. In the analyses of short-term emissions resulting from an incinerator scrubber failure, the mixing of incompatible wastes, and abnormal pH conditions during cyanide treatment or sulfide treatment, potential health risks were evaluated by comparing the predicted off-site concentrations in air to a concentration-based acute no-observed-effect-level (NOEL). This procedure was used since the exposure evaluated would be expected to cause a localized irritating effect. Therefore, estimates of the systemic dose were not required. For the tank farm fire, however, the dose received over estimated period of exposure was calculated since the surrogate chemical, methylene chloride, could produce systemic effects. The following equation was used to calculate the dose received:

TABLE I-19

DURATION OF EXPOSURE FOR SHORT-TERM EVENTS

Source of Release	Chemical of Concern	Duration of Exposure
Tank Farm Fire	Methylene chloride	2.6 h maximum
Mixing of Incompatible Wastes	Hydrogen sulfide	42 min
Incinerator Scrubber Failure	Sulfur dioxide Nitrogen oxides Hydrogen chloride Hydrogen fluoride	30 sec
Cyanide Treatment Failure	Hydrogen cyanide	NA
Sulfide Treatment Failure	Hydrogen sulfide	NA

NA: Not available, but assumed to be brief.

D_f = (Ca)(Ia)(t)(AF)/(BW)

Where:

D_f = Human dose, mg/kg

Ca = Contaminant concentration in air, mg/m^3

Ia = Breathing rate, m^3/h

t = Exposure time, h

AF = Absorption factor, dimensionless

BW = Body weight, kg

The following assumptions were made in estimating the human dose:

- Ia = 1.7 m^3/h, based on an average male performing light work
- t = 2.6 h (Monenco 1985)

I.3.3 OFF-SITE TRANSPORTATION SPILLS

Short-term exposures through inhalation of air contaminated as a result of a transportation spill onto a roadway were calculated using the following equation:

D_{tr} = (Ca)(Ia)(t)(AF)/(BW)

Where:

D_{tr} = Human dose, mg/kg

C_a = Contaminant concentration in air, mg/m^3

Ia = Inhalation rate, m^3/h

t = Exposure time, h

AF = Absorption factor, dimensionless

BW = Body weight, kg

The following assumptions were made in estimating the dose:

- Ia = 1.7 m^3/h based on an adult performing light work (Altman and Dittmer 1974); 0.78 m^3/h for a child during light activity
- t = 1 hour, based on the predicted emergency response time on the local access route. This represents the period of time estimated to be necessary to prevent further human exposures.
- BW = 70 kg for an adult (NIOSH 1980); 31 kg for a child (ICRP 1984)

Short-term exposures associated with drinking water contaminated as a result of a transportation spill into a stream were calculated using the following equation:

$$D_{tw} = (Cw)(Iw)(AF)/(BW)$$

Where:

D_{tw} = Human dose, mg/kg

Cw = Contaminant concentration in water, mg/l

Iw = Ingestion rate of water, l

AF = Absorption factor, dimensionless

BW = Body weight, kg

Although ingestion was evaluated as the pathway for exposure in the risk assessment, no drinking water intakes have been reported along the length of Twenty Mile Creek downstream from the access road bridge. For this reason, it has been assumed in the risk assessment that an individual located at the edge of Twenty Mile Creek takes water directly from the stream. The amount of water ingested by this individual has been assumed to be one-fourth of the average daily consumption of water; 0.5 l for an adult (USEPA 1980) or 0.25 l for a child (USEPA 1980). The assumed body weight for a child was 31 kg; for an adult, 70 kg.

I.3.4 SUMMARY OF HUMAN DOSES ASSOCIATED WITH SHORT-TERM RELEASES

The predicted human doses associated with the tank farm fire and the off-site transportation accidents are summarized in Tables I-20 and I-21 for adults and children, respectively. The chemical concentrations in air and stream water were derived using the methods presented in Appendices D and E, respectively.

TABLE I-20

PREDICTED HUMAN DOSES ASSOCIATED WITH SHORT-TERM EXPOSURES (ADULTS)

Source of Release	Chemical of Concern	Environmental Concentration		Dose* (mg/kg)
Tank Farm Fire	Methylene Chloride	1 060 (maximum)	mg/m^3	67
Spill onto the Roadway	Methylene Chloride	18 400	mg/m^3	447
Spill into the Stream	Methylene Chloride	9.6	mg/l	0.069
	Pyridine			
	• 230 liter spill			
	– 100 m downstream	541	mg/l	3.9
	– 3 000 m downstream	99	mg/l	0.71
	– 6 400 m downstream	68	mg/l	0.49
	• 1 500 liter spill			
	– 100 m downstream	3 609	mg/l	25.8
	– 3 000 m downstream	659	mg/l	4.7
	– 6 400 m downstream	451	mg/l	3.2
	• 19 000 liter spill			
	– 100 m downstream	45 112	mg/l	322
	– 3 000 m downstream	8 236	mg/l	58.8
	– 5 400 m downstream	5 639	mg/l	40.3

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* For spills into the stream, the dose is based on ingestion of 0.5 liter of water by a 70 kg adult. For atmospheric emissions (spill onto a roadway and a tank farm fire), the dose is based on the predicted period of exposure and a breathing rate of 1.7 m^3/h.

TABLE I-21

PREDICTED HUMAN DOSES ASSOCIATED WITH SHORT-TERM EXPOSURES (CHILDREN)

Source of Release	Chemical of Concern	Environmental Concentration		Dose* (mg/kg)
Tank Farm Fire	Methylene Chloride	1 060 (maximum)	mg/m^3	69
Spill onto the Roadway	Methylene Chloride	18 400	mg/m^3	463
Spill into the Stream	Methylene Chloride	9.6	mg/l	0.077
	Pyridine			
	• 230 liter spill			
	– 100 m downstream	541	mg/l	4.4
	– 3 000 m downstream	99	mg/l	0.80
	– 6 400 m downstream	68	mg/l	0.55
	• 1 500 liter spill			
	– 100 m downstream	3 609	mg/l	29.1
	– 3 000 m downstream	659	mg/l	5.3
	– 6 400 m downstream	451	mg/l	3.6
	• 19 900 liter spill			
	– 100 m downstream	45 112	mg/l	363.8
	– 3 000 m downstream	8 236	mg/l	66.5
	– 5 400 m downstream	5 639	mg/l	45.5

---

* For spills into the stream, the dose is based on ingestion of 0.25 liter of water by a 31 kg child. For atmospheric emissions (spill onto a roadway and a tank farm fire), the dose is based on the predicted period of exposure and a breathing rate of 0.78 m^3/h.

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APPENDIX J

CALCULATION OF AVERAGE DAILY SYSTEMIC DOSES IN THE ENVIRONMENTAL RISK ASSESSMENT

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APPENDIX J

CALCULATION OF AVERAGE DAILY SYSTEMIC DOSES IN THE ENVIRONMENTAL RISK ASSESSMENT

J.1 INTRODUCTION

As discussed in Chapter 11 of this report, the final step in the exposure assessment for environmental species is the calculation of systemic dose. In this appendix, the major assumptions used in estimating the contaminant dose received by the animal species considered in the analysis are discussed. The equations used in calculating the systemic dose resulting from exposures to facility emissions are also presented.

J.2 ASSUMPTIONS REGARDING LEVELS OF CONTAMINANTS IN THE DIET

Defining the dietary patterns of wildlife and livestock species is exceedingly complex. For wildlife, in particular, diets are not only poorly characterized in the literature, but also change from season to season and from region to region. Furthermore, estimating the potential contaminant levels in specific components of the diet (e.g., nuts, tree bark, or insects) is subject to considerable uncertainty. Therefore, simplifying assumptions were made in the current environmental risk assessment concerning the composition of the diet for indicator species and the concentrations of chemicals of concern in the components of the diet. These assumptions are summarized in Table J-1.

To estimate the concentrations of chemicals of concern in herbivorous diets, concentration estimates in crops derived in the human health risk assessment were used. Hay was selected as the crop estimated to contain the highest levels of chemicals of concern. It was conservatively assumed that chemicals of concern were present in hay at the maximum annual concentrations predicted by the modeling of emissions from the facility.

Similarly, the diets of carnivores (other than the heron) were assumed to contain chemicals of concern at the maximum annual concentrations in beef predicted in the human health risk assessment. For herons, the diet was assumed to consist entirely of fish from the Welland River. The concentrations of chemicals in fish were based on the predicted levels of contamination in the Welland River and application of

TABLE J-1

ASSUMPTIONS REGARDING FOOD AND WATER CONSUMPTION PATTERNS

Species[a]	Drinking Water Source	Dietary Composition
Herbivores	River[b]	100% vegetation[c]
Carnivores	River	100% meat[d]
Omnivores	River	50% vegetation, 50% meat

---

[a] See Table J-2 for designation of indicator species as herbivores, carnivores, or omnivores.

[b] River concentrations are those derived for the Welland River.

[c] It was assumed that the chemicals of concern were present in hay at the maximum annual concentrations, as derived in the human health risk assessment. Of the crops modeled in the risk assessment, hay is the crop estimated to contain the highest levels of contaminants.

[d] Except for herons, it was assumed that the chemicals of concern were present at the maximum annual concentrations in beef, as derived for purposes of the human health risk assessment. For herons, the diet was assumed to consist of fish from the Welland River.

bioconcentration factors (BCFs). (These bioconcentration factors are presented in Appendix L.) For omnivores, diets were assumed to consist of 50 percent vegetation and 50 percent meat, using a composite of concentrations in hay and beef as defined above.

For all species considered in the environmental risk assessment, the drinking water supply was assumed to be taken from the Welland River. This was considered a conservative approach, since, in general, contaminant concentrations were calculated to be higher in the Welland River than in other potential drinking water sources, e.g., ponds and cisterns.

J.3 CALCULATION OF SYSTEMIC DOSE

For livestock, mammalian wildlife, and avian species, predicted exposure levels and safe levels of exposure were expressed as systemic doses in units of mg chemical/kg body weight/day (mg/kg/day). The calculation of systemic dose was undertaken using standard dietary and metabolic assumptions for the indicator species. The equations used to derive systemic doses based on chronic inhalation, ingestion of drinking water, ingestion of feed, ingestion of soil, or ingestion of fish are presented below. Procedures used to estimate acute inhalation exposures are also presented. The physiologic constants for the various indicator species are contained in Table J-2.

J.3.1 CHRONIC INHALATION EXPOSURE

Chronic exposures through inhalation were calculated using the following equation:

$$D_i = \frac{Ca\ Va}{W}$$

Where:

D_i = systemic dose via inhalation, mg/(kg-d)

Ca = concentration in air, mg/m^3

Va = respiratory volume, m^3/d

W = body weight, kg

For concentration data reported in volumetric units, i.e., parts per million (ppm), the following relationship was used to convert mass units, i.e., mg/m^3:

$$Ca,m = \frac{Ca,v\ M}{1000\ k}$$

Where:

Ca,m = mass concentration of substance in air, mg/m^3

Ca,v = volumetric concentration of substance in air, ppm

M = molecular weight of substance, g/mole

k = molar volume of ideal gas, 0.02445 m^3/mole (25°C, 760 mm Hg)

TABLE J-2

STANDARD VALUES USED FOR DOSE CONVERSIONS FOR LIVESTOCK/WILDLIFE INDICATOR SPECIES

Species	Type	Carnivore Herbivore Omnivore	Body Weight (kg)	Respiratory Volume (m^3/day)	Water Consumption (L/day)	Food Consumption (kg/day)	Soil Ingestion (kg/day)
Calf	dm	h	200 (1)	30 (1)	15 (1)	5 (1)	0.3 (n)
Chicken (leghorn)	db	h	1 (d,f)	1 (1)	0.2 (e,f)	0.07 (e)	---
Dairy cow	dm	h	450 (f)	125 (1)	60 (f)	17 (n)	1.1 (n)
Duck	nb	o	1.5 (o)	1.5 (p)	0.3 (p)	0.15 (o)	---
Goat	dm	h	60 (1)	7 (1)	8 (1)	2 (1)	0.09(u)
Guinea pig	lm	h	0.5 (a)	0.3 (d)	0.085 (a)	0.03 (a)	---
Hamster	lm	h	0.125 (a)	0.12 (d)	0.015 (b)	0.015 (a)	---
Heron	nb	c	3 (i)	3 (o)	0.6 (o)	0.3 (j,k)	---
Horse (light work)	dm	h	450 (f)	104 (1)	30 (f)	8 (d,f)	0.4 (u)
Mink	lm	c	1 (m)	1 (p)	0.3 (p)	0.15 (m)	---
Mouse	lm	o	0.025 (a)	0.042 (d)	0.005 (a)	0.003 (a)	---
Pig	dm	h	100 (d,f)	8 (1)	8 (f)	2.5 (a,d)	0.2 (s)
Quail	nb	h	0.17 (h)	0.17 (o)	0.033 (o)	0.015 (h)	---
Rabbit	nm	h	2 (a)	1.5 (c)	0.33 (a)	0.06 (a)	---
Rat	lm	o	0.25 (a)	0.15 (d)	0.025 (a)	0.015 (a)	---
Sheep	[illegible]	h	70 (d,f)	9 (1)	6 (f)	2 (f)	0.09(t)
Steer	[illegible]	h	500 (d)	125 (1)	40 (f)	9 (n)	0.7 (n)

db - domestic bird
dm - domestic mammal
lm - laboratory mammal
nb - native bird
nm - native mammal

(a) NIOSH 1980
(b) Hoeltge, Inc.
(c) Spector 1956
(d) Altman and Dittmer 1974
(e) Ecologistics 1984
(f) NRC 1974
(g) NRC 1978
(h) USEPA 1986
(i) Palmer 1962
(j) Patuxent Wildlife Research Center 1986
(k) Martin et al. 1951
(l) Ecologistics 1986 (assuming 6% of dry matter intake)
(m) USEPA 1980j
(n) Ecologistics 1986
(o) By analogy to data for herons
(p) By analogy to data for chickens
(q) Estimated from data for other mammals
(r) Average daily consumption of fish based on assumption that daily food consumption is equivalent to 15-17% body weight and that 68% of the diet consists of fish.
(s) Fries et al. 1982 (assuming 8% of dry matter intake)
(t) Fries 1982 (assuming 4.5% of dry matter intake)
(u) Estimated from data for sheep

J.3.2 EXPOSURE VIA INGESTION OF DRINKING WATER

Chronic exposures through the ingestion of water were calculated using the following equation:

$$Dw = \frac{Cw\ Vw}{W}$$

Where:

Dw = systemic dose via drinking water, mg/(kg-d)

Cw = concentration in drinking water, mg/l or ppm

Vw = volume of water consumed per day, l/day

W = body weight, kg

J.3.3 EXPOSURE VIA INJESTION OF FEED

Chronic exposures through ingestion of feed were calculated using the following equation:

$$Df = \frac{Cf\ Wf}{W}$$

Where:

Df = systemic dose via ingestion of feed, mg/(kg-d)

Cf = concentration in feed, mg/kg

Wf = weight of food consumed per day, kg/d

W = body weight, kg

J.3.4 EXPOSURE VIA INGESTION OF SOIL (DAIRY COW, STEER, PIG, AND SHEEP ONLY)

Chronic exposures through the ingestion of soil were calculated using the following equation:

$$Ds = \frac{Cs\ Ws}{W}$$

Where:

Ds = systemic dose via ingestion of soil, mg/(kg-d)

Cs = concentration in soil, mg/kg

Ws = weight of soil consumed per day, kg/d

W = body weight, kg

J.3.5 EXPOSURE VIA INGESTION OF FISH (HERON ONLY)

Chronic exposures through the ingestion of contaminated fish were calculated for the heron using the following equation:

$$Da = \frac{Cw\ BCF\ Wf}{W}$$

Where:

Da = systemic dose via ingestion of fish, mg/(kg-d)

Cw = concentration in water, mg/L or ppm

BCF = bioconcentration factor for freshwater fish species, (mg/kg)/(mg/L)

Wf = weight of fish consumed per day, kg/d

W = body weight, kg

The BCF values used in the risk assessment are shown in Table J-3.

J.3.6 TOTAL SYSTEMIC DOSE

The total chronic systemic dose was calculated by summing the systemic doses received from inhalation of chemicals in air, ingestion of chemicals in drinking water, ingestion of

TABLE J-3

BIOCONCENTRATION FACTORS FOR FISH

Chemical	BCF[1]	Reference
Acetone	0.1	Calculated[2]
Acrylonitrile	48	USEPA 1980a
Arsenic	4	USEPA 1980b
Benzene	13	Calculated[2]
Benzo(a)Anthracene	930	By analogy[4]
Benzo(a)Pyrene	930	USEPA 1980k
Beryllium	19	USEPA 1980c
Bromodichloromethane	12	Calculated[2]
Bromoform	18	Calculated[2]
Butadiene	10	Calculated[2]
Cadmium	2 213	USEPA 1985a
Carbon tetrachloride	30	USEPA 1984
Chlorobenzene	450	USEPA 1985b
Chloroform	6	USEPA 1980d
Chromium	1	USEPA 1980e
Copper	220	Callahan *et al*. 1979
Dichlorodifluoromethane	1	Default value[3]
Ethanol	0.1	Calculated[2]
Ethylene glycol	0.005	Calculated[2]
Ethylene oxide	0.34	USEPA 1985e
Formaldehyde	0.06	Calculated[2]
Hexachlorobenzene	44 437	USEPA 1985b
Hydrazine	0.01	Calculated[2]
Hydrogen chloride	1	Default value[3]
Hydrogen cyanide	0	USEPA 1980f, 1985c
Hydrogen fluoride	2	NRCC 1977
Hydrogen sulfide	1	Default value[3]
Lead	45	USEPA 1985d
Mercury	63 000	USEPA 1980g
Methyl ethyl ketone	0.3	Calculated[2]
Methylene chloride	2	Calculated[2]
Naphthalene	427	Veith *et al*.1979
Nickel	61	USEPA 1980h
Nitrogen oxides	1	Default value[3]
0-Nitrophenol	6	Calculated[2]
PCBs	10 400	USEPA 1980j
PCDD	26 000	Adams *et al*. 1987
Petroleum distillates	0.7	Calculated[2]
Phenol	2	USEPA 1980i
Pyridine	0.7	Calculated[2]
Styrene	64	Calculated[2]
Sulfur dioxide	1	Default value[3]

TABLE J-3 (continued)

BIOCONCENTRATION FACTORS FOR FISH

Chemical	BCF[a]	Reference
Tetrachloroethylene	49	USEPA 1980l
Tetraethyl lead	130	Verschueren 1983
Toluene	39	Calculated[2]
Trichloroethane	9	USEPA 1981
Trichloroethylene	17	USEPA 1980m
Trichlorotrifluoroethane	1	Default value[3]
Xylene	83	Calculated[2]
Zinc	432	USEPA 1980n

---

[1] Bioconcentration factors are those for freshwater fish species only.

[2] Calculated according to the regression equation as follows (Veith *et al*. 1979):

$$\log \text{BCF} = (0.85 \log K_{ow}) - 0.7$$

Where BCF = bioconcentration factor

K_{ow} = octanol-water partition coefficient

[3] No BCF or octanol-water partition coefficient was available. A default value of 1 was used.

[4] Estimated by analogy to benzo(a)pyrene. This value may overestimate the BCF for benzo(a)anthracene, since studies demonstrate that most polynuclear aromatic hydrocarbons have low tendency to bioaccumulate (USEPA 1980k).

chemicals in feed or vegetation, and ingestion of chemicals in soil. The following equation was used in the risk assessment:

$$\text{Total systemic dose} = Di + Dw + Df + Ds$$

Where:

Di = systemic dose via inhalation of chemicals in air, mg/(kg-d)

Dw = systemic dose via ingestion of chemicals in drinking water, mg/(kg-d)

Df = systemic dose via ingestion of chemicals in feed, mg/(kg-d)

Ds = systemic dose via ingestion of chemicals in soil, mg/(kg-d)

In the case of the heron, the dose received via ingestion of chemicals in fish (Da), rather than the dose received via ingestion of feed (Df), was included in the calculation.

J.3.7 ACUTE INHALATION EXPOSURE

Acute inhalation exposures were calculated similarly to chronic inhalation exposure, using the following equation:

$$Dai = \frac{Ca\ Va\ f}{W}$$

Where:

Dai = acute systemic dose via inhalation, mg/(kg-d)

Ca = concentration in air, mg/m^3

Va = respiratory volume, m^3/d

f = adjustment factor, to adjust for less than continuous daily exposure (e.g., for an exposure received two hours/day, f = 2/24 = 0.083)

W = body weight, kg

Expression of doses as average doses over a day (24-hour period) permitted comparison with the criterion doses, which were based on exposures of varying durations also averaged over a 24-hour period by the application of adjustment factors.

For concentration data reported in volumetric units, i.e., parts per million (ppm), the relationship presented in Section J.3.1 was used.

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APPENDIX K

USE OF SAFETY FACTORS TO DERIVE SAFE EXPOSURE LEVELS IN THE ENVIRONMENTAL RISK ASSESSMENT

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APPENDIX K

USE OF SAFETY FACTORS TO DERIVE SAFE EXPOSURE LEVELS IN THE ENVIRONMENTAL RISK ASSESSMENT

K.1 INTRODUCTION

Safety factors were developed in the environmental risk assessment to derive safe exposure levels based on available toxicity data. Safety factors are used to account for:

- Limitations in the available toxicity data,
- Differences between the conditions of exposure under which the toxicity data were collected and conditions of exposure for species in the field,
- Differential sensitivity to toxicants among environmental species,
- Intraspecies variation in sensitivity, and
- Extrapolation of effects on a single species to a population or ecosystem.

This appendix provides a description of the approaches used to develop the safety factors applied in the environmental risk assessment.

K.2 ACUTE TOXICITY

In assessments of ecological risk associated with acute toxicity (or exposure to sudden, upset conditions), the acute exposure level for a species should be based on a no-observed-adverse-effect level (NOAEL) for acute, short-term exposures. However, lethality often serves as the response upon which acceptable short-term exposure limits are based, primarily because it is the only type of data available. Where lethality data (e.g., LD_{50} or LC_{50} values*) or data providing a lowest-observed-adverse- effect level (LOAEL) are the only acute toxicity data available, these data must be used to estimate exposure levels associated with no significant adverse effects in the species tested.

EPA's Office of Pesticide Programs (USEPA 1986) has established regulatory risk criteria for acute toxicity, in which a "presumption of no risk" is reached if the estimated environmental concentration or dose does not exceed one-fifth the LC_{50} or LD_{50} for mammals and birds or one-tenth the LC_{50} for aquatic organisms. The safety factor of 5 was derived by probit analysis of dose-response relationships for test populations of varying sensitivity. Probit analysis indicated that a dose 5 times lower than the LD_{50} or LC_{50} would be expected to lead to a mortality rate of about 0.1 percent for typical populations of fish and wildlife species, and to a mortality rate of about 10% for populations of highly variable toxicant sensitivity. Given EPA's regulatory risk criteria and accepting the assumptions of the probit model for analyzing acute toxicity data, it can be interpreted that 0.1% lethality (or one death in 1000) of the typical population exposed to an environmental contaminant is an insignificant or acceptable response in a population. Using this precedent, a safety factor of five was applied to acute LD_{50} or LC_{50} data to derive a safe exposure level for acute effects for the species tested. In those cases where a LOAEL was available, an uncertainty factor of half of five, or 2.5, was applied to derive an acute safe exposure level.

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* LD_{50} and LC_{50}: the median lethal dose (or concentration), or the dose (or concentration) of chemical needed to produce death in 50% of the exposed animals.

The safety factors used in the OWMC environmental risk assessment are summarized in Table K-1.

K.3 CHRONIC TOXICITY

Chronic toxicity data tend to be more limited than acute toxicity data for agricultural and wildlife species. Therefore,

TABLE K-1

SAFETY FACTORS USED IN PREDICTING ACUTE SAFE EXPOSURE LEVELS FOR ENVIRONMENTAL RECEPTORS

Available Data	Safety Factor*
Acute NOAEL	1
Acute LOAEL (other than lethality)	2.5
Lethality	5

---

* An additional safety factor of 10 was applied to the safe systemic dose or ambient concentration for a species to account for limitations in the available toxicologic data (see Sources of Additional Uncertainty). The criterion doses or concentrations incorporate this additional 10-fold safety factor.

a chronic NOAEL may have to be derived by application of an safety factor to acute or subchronic toxicity data.

Unlike human health risk assessment, few precedents exist for the quantitative prediction of a chronic NOAEL from acute or subchronic toxicity data. As stated by Slooff et al. (1986), "from a theoretical point of view, acute toxicity tests with death as the endpoint can never predict changes in reproduction, behavior, physiology or biochemistry resulting from chronic exposure since the biological basis for lethal and non-lethal responses may be quite different." Empirical data exist, however, that justify the prediction of chronic toxicity from acute toxicity, including lethality. Slooff et al. (1986) conducted a correlation and regression analysis on corresponding literature data for acute and chronic sensitivities of fish and daphnids for 164 chemical compounds. They determined the relationship of acute versus chronic toxicity to be:

$$\log \text{NOEC} = -1.28 + 0.95 \log \text{L(E)C}_{50}$$

Where:

NOEC = no-observed-effect concentration

L(E)C_{50} = LC_{50} or EC_{50} = concentration producing lethality or some other adverse effect in 50% of the population

Using this regression equation, at an LC_{50} (or EC_{50}) of 1 mg/L, the NOEC is calculated to be 0.05 mg/L, or one-twentieth of the acute value. Applying an additional "uncertainty factor" of 25 (based on the lower confidence limit reported by Slooff et al. for the regression equation), the overall safety factor for extrapolating from acute lethality data to an NOEC is 20 x 25 or 500. In other words, application of a safety factor of 500 to acute lethality data will predict an NOEC that is equal to or smaller than an experimentally derived NOEC 95 percent of the time.

A similar analysis was conducted by Kenaga (1982) to assess the predictability of chronic toxicity from acute toxicity of chemicals in aquatic species. The relationship of acute to chronic toxicity was determined as the inverse of the ratio of the acute LC_{50} to the chronic no-observed-effect concentration (NOEC), for the same species and was expressed as the acute/chronic ratio (ACR). The analysis was based on data for 84 different chemicals (including pesticides, inorganic chemicals, and industrial chemicals) and nine species of fish and two species of aquatic invertebrates. Kenaga found that 86 percent of the LC_{50} data were less than 100-fold different from the chronic NOEC for the same chemicals and species and 92% were less than 500-fold different. Among the industrial organic chemicals (i.e., excluding pesticides and metals), the average ACR for four aquatic species was 12; 93 percent of the ACR values were 25 or below. The differences between acute and chronic toxicity values were greater for pesticides and heavy metals. The results of the Kenaga analysis support the findings of Slooff et al. (1986) that for most chemicals, the ratio of acute to chronic toxicity is no greater than 500-fold, and in fact is generally no greater than 25.

Based on the above analyses, a safety factor of 500 was used in the environmental risk assessment for predicting safe chronic exposure levels from acute lethality data (i.e., LD_{50} or LC_{50} data) for a given species. It should be recognized that the Slooff et al. analysis was limited to aquatic life and does not necessarily apply to other types of wildlife. However, because similar empirical data do not exist for other species, a safety factor of 500 was used for acute to chronic toxicity extrapolations for all environmental receptors. Where a chronic NOAEL must be derived from acute NOAEL or acute LOAEL data, safety factors of 100 and 250, respectively, were applied.

In the absence of chronic toxicity data, subchronic toxicity data may also serve as the basis for deriving chronic safe exposure levels. In human risk assessments, a 10-fold safety factor is traditionally used for extrapolating from subchronic to chronic exposure. Additional safety factors of between 1 and 10 are also applied in human risk assessment when deriving a NOAEL from a LOAEL (Dourson and Stara 1983). In the absence of experimental data in support of safety factors specific to environmental species, these guidelines for the use of safety factors from human risk assessment were applied in the environmental risk assessment.

The safety factors used to derive safe chronic exposure levels are summarized in Table K-2.

K.4 SOURCES OF ADDITIONAL UNCERTAINTY

Additional uncertainty in the derivation of safe exposure levels for environmental receptors is introduced by various limitations in the available experimental data. These uncertainties result from factors such as:

- Differences in sensitivity to toxicants for different life stages and sexes of the same species,
- Differences in sensitivity to toxicants among environmental species,
- availability only of experimental data for higher order effects such as mortality that do not reflect the more subtle and sensitive endpoints that also serve as indicators of species survival (e.g., enzymatic and behavioral changes),
- variations in the toxicity observed under laboratory and field conditions, and
- extrapolation of effects on a single species to a population or ecosystem.

The effect of these uncertainties on the assessment of environmental risk, however, cannot be quantified. Lack of

TABLE K-2

SAFETY FACTORS USED IN PREDICTING CHRONIC SAFE EXPOSURE LEVELS FOR ENVIRONMENTAL RECEPTORS

Available Data	Safety Factor*
Chronic	
NOAEL	1
LOAEL (other than lethality)	3
Lethality	10
Subchronic	
NOAEL	10
LOAEL (other than lethality)	30
Lethality	100
Acute	
NOAEL	100
LOAEL (other than lethality)	250
Lethality	500

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* An additional safety factor of 10 was applied to the safe systemic dose or ambient concentration to account for limitations in the available toxicologic data (see Sources of Additional Uncertainty). The criterion doses and concentrations incorporate this additional safety factor.

knowledge of how these factors alter the toxic response of various species to any single surrogate chemical could result in either an overestimate or underestimate of risk. In the absence of any empirical data to quantify the level of uncertainty, an additional safety factor of 10 was applied to the experimental NOAEL or LOAEL in order to derive safe acute and chronic exposure levels.

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U.S. Environmental Protection Agency (USEPA). 1986. Ecological Risk Assessment. Hazard Evaluation Division, Standard Evaluation Procedure. Office of Pesticide Programs, Washington, D.C. EPA-540/9-85-001.

APPENDIX L

DERIVATION OF CRITERION DOSES AND CONCENTRATIONS IN THE ENVIRONMENTAL RISK ASSESSMENT

TABLES

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L-2 Criterion Doses and Concentrations for Chronic Exposure L-10

REFERENCES L-14

TABLE L-1

DOCUMENTATION OF ENVIRONMENTAL TOXICITY DATA

TABLE L-1

Documentation of Environmental Toxicity Data 1

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)		Tested Species	Notes
Mac & Seelye 81	acetone	fish	no effects	aq	chronic	NOEL	1.000	7.9E+00	mg/L	1	7.90E+00	mg/L	trout	
Bruckner & Peters	acetone	rat	intoxication	inh	chronic	LOEL	0.125	1.9E+04	ppm	30	1.15E+02			
EPA 1983	acrylonitrile	fish	LC50	aq	chronic	LOEL	1.000	2.2E+00	mg/L	10	2.20E-01	mg/L	trout	
EPA 1983	acrylonitrile	rabbit	wt. loss	inh	chronic	LOEL	0.119	1.0E+02	ppm	30	6.58E-01			
EPA 1983	acrylonitrile	rat	reproduction	gav	chronic	NOEL	1.000	2.5E+01	mg/kg/day	10	2.50E+00			
EPA 1983	acrylonitrile	rat	death	ingw	chronic	LOEL	1.000	3.5E+01	ppm	3	1.17E+00			
Ecologistics 86b	arsenic	chicken	growth/eggs	ingf	chronic	LOEL	1.000	5.0E+00	ppm	30	1.17E-02			
NRC 1980a	arsenic	dairy cow	no effect	ingf	chronic	NOEL	1.000	4.7E+00	mg/kg/day	1	4.68E+00			Pb arsenate(discrepancy in
EPA 1981	arsenic	fish		aq	chronic	LOEL	1.000	8.9E-01	mg/L	3	2.97E-01	mg/L	fathead	
EPA 1981	arsenic	fish	phys. alt.	aq	chronic	LOEL	1.000	3.0E-01	mg/L	3	1.00E-01	mg/L	salmon	
Ecologistics 86b	arsenic	horse		ingf	chronic	NOEL	1.000	5.3E-01	mg/kg/day	1	5.30E-01			
EPA 1981	arsenic	plant2	growth	soil	chronic	LOEL	1.000	2.5E+01	ppm	3	8.33E+00	ppm		
Ecologistics 86a	arsenic	plant2	growth	soil	chronic	LOEL	1.000	2.2E+01	ppm	3	7.33E+00	ppm	corn	
EPA 1981	arsenic	rat	weight	ingf	chronic	LOEL	1.000	1.3E+01	mg/kg/day	3	4.17E+00			
Ecologistics 86b	arsenic	sheep		ingf	chronic	NOEL	1.000	1.7E+01	ppm	1	4.89E-01			
EPA 1980	benzene	fish	LC50/EC50	aq	chronic	LOEL	1.000	5.3E+00	mg/L	500	1.06E-02	mg/L	trout	
EPA 1984g	benzene	mouse	reproduction	gav	chronic	LOEL	1.000	3.0E-01	mg/kg/day	30	1.00E-02			
Ecologistics 86b	benzene	pig	blood tox.	inh	chronic	NOEL	0.179	2.0E+01	ppm	10	9.30E-02			
Ecologistics 85	benzene	plant1	spots	air	chronic	LOEL	1.000	3.0E+01	mg/m3	250	1.20E-01	mg/m3	bean	
Ecologistics 86b	benzene	rabbit	leukopenia	gav	chronic	LOEL	1.000	1.0E+00	mg/kg/day	3	3.33E-01			
EPA 1984g	benzene	rat	leukopenia	inh	chronic	NOEL	0.208	3.1E+01	ppm	1	1.26E+01			
A 1984g	benzene	rat	leukopenia	gav	chronic	NOEL	1.000	7.1E-01	mg/kg/day	1	7.10E-01			
PA 1980m	benzo(a)anthracene	fish	mortality	aq	chronic	LOEL	1.000	1.0E+00	mg/L	500	2.00E-03	mg/L	bluegill	
olfe & Bryan 39	benzo(a)anthracene	rat	reproduction	inj	chronic	LOEL	1.000	2.0E+01	mg/kg/day	30	6.67E-01			
ARC 1983	benzo(a)pyrene	mouse	reproduction	gav	chronic	LOEL	1.000	1.0E+01	mg/kg/day	30	3.33E-01			
EPA 1980a	beryllium	fish	LC50/EC50	aq	chronic	LOEL	1.000	3.3E+00	mg/L	500	6.50E-03	mg/L	fathead	
EPA 1980a	beryllium	fish	LC50/EC50	aq	chronic	LOEL	1.000	1.3E+00	mg/L	500	2.60E-03	mg/L	bluegill	
Yopp 1974	beryllium	plant2	growth	soil	chronic	LOEL	1.000	1.0E+00	ppm	3	3.33E-01	ppm	corn	
EPA 1983a	beryllium	plant2	yield	soil	chronic	LOEL	1.000	8.0E+01	mg/kg	3	2.67E+01	ppm	lettuce	
EPA 1984e	beryllium	rat	lung	inh	chronic	LOEL	1.000	3.4E-02	mg/m3	3	6.80E-03			
EPA 1984e	beryllium	rat		ingf	chronic	NOEL	1.000	5.0E+01	mg/kg/day	1	5.00E+01			
EPA 1980f	bromodichloromethane	mouse	immune depr.	gav	chronic	LOEL	1.000	1.3E+02	mg/kg/day	3	4.17E+01			
EPA 1980f	bromodichloromethane	mouse	reproduction	inh	chronic	LOEL	1.000	8.4E+03	mg/m3	30	4.69E+02			
EPA 1980f	bromodichloromethane	mouse	dec. water cons.	ingw	chronic	LOEL	1.000	3.0E+02	mg/L	3	2.00E+01			
EPA 1980f	bromoform	fish	LC50	aq	chronic	LOEL	1.000	2.9E+01	mg/L	500	5.86E-02	mg/L	bluegill	

Documentation of Environmental Toxicity Data 2

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)		Tested Species	Notes
EPA 1980f	bromoform	mouse	immune depr.	gav	chronic	LOEL	1.000	1.3E+02	mg/kg/day	3	4.17E+01			
EPA 1980f	bromoform	rat	liver, renal	inh	chronic	NOEL	1.000	5.0E+01	mg/m3	10	3.00E+00			
Verschueren 1983	butadiene	fish	TLm	aq	chronic	LOEL	1.000	7.2E+01	mg/L	500	1.43E-01	mg/L	pinperch	
Clayton 1981	butadiene	rat	infiltration	gav	chronic	LOEL	1.000	1.0E+02	mg/kg/day	10	1.00E+01			
NTP 1984	butadiene	rat	reproduction	inh	chronic	LOEL	0.250	2.0E+02	ppm	30	2.25E+00			
Ecologistics 86b	cadmium	chicken	eggs	ingf	chronic	NOEL	1.000	3.0E+00	ppm	1	2.10E-01			
Ecologistics 86b	cadmium	dairy cow	reproduction	ingf	chronic	LOEL	1.000	5.0E+01	ppm	3	6.67E-01			
DiGialio 1984	cadmium	duck	body wt.	ingf	chronic	LOEL	1.000	4.5E+02	ppm	30	1.50E+00			
EPA 1984	cadmium	fish		aq	chronic	LOEL	1.000	2.0E-03	mg/L	3	6.67E-04	mg/L	salmon	
EPA 1984	cadmium	fish		aq	chronic	LOEL	1.000	7.0E-03	mg/L	3	2.33E-03	mg/L	sucker	
Yopp 1974	cadmium	plant2	growth	soil	chronic	LOEL	1.000	2.5E+00	ppm	3	8.33E-01	ppm	soybean,etc.	
Bar.&Sull. 1982	cadmium	rat	reproduction	gav	chronic	LOEL	1.000	8.0E+00	mg/kg/day	3	2.67E+00			
NAS 1982	cadmium	rat	kidney	ingw	chronic	NOEL	1.000	7.5E-01	mg/kg/day	1	7.50E-01			
EPA 1984d	carbon tetrachloride	fish	no effects	aq	chronic	NOEL	1.000	3.4E+00	mg/L	1	3.40E+00	mg/L	minnow	
EPA 1984d	carbon tetrachloride	guinea pig	liver	inh	chronic	NOEL	1.000	6.1E+00	mg/m3	1	3.66E+00			
EPA 1984d	carbon tetrachloride	rat	no effects	ingf	chronic	NOEL	1.000	1.0E+01	mg/kg/day	1	1.00E+01			
EPA 1984d	carbon tetrachloride	rat	liver	inh	chronic	NOEL	1.000	6.1E+00	mg/m3	1	3.66E+00			
EPA 1985c	chlorobenzene	fish	equilibrium loss	aq	chronic	LOEL	1.000	2.1E+00	mg/L	3	7.00E-01	mg/L	trout	
EPA 1985c	chlorobenzene	mouse	survival	gav	chronic	LOEL	0.714	3.0E+01	mg/kg/day	3	7.14E+00			
EPA 1985c	chlorobenzene	rat	liver, kidney	inh	chronic	NOEL	0.208	7.5E+01	ppm	1	6.40E+01			
EPA 1980c	chloroform	fish	avoidance	aq	chronic	LOEL	1.000	1.5E+02	mg/L	3	4.94E+01	mg/L	stickleback	
EPA 1980c	chloroform	fish	LC50	aq	chronic	LOEL	1.000	1.2E+00	mg/L	10	1.24E-01	mg/L	trout	
EPA 1985b	chloroform	mouse	kidney	gav	chronic	LOEL	0.857	6.0E+01	mg/kg/day	3	1.71E+01			
EPA 1985b	chloroform	rat	liver	gav	chronic	LOEL	0.857	6.0E+01	mg/kg/day	3	1.71E+01			
EPA 1985b	chloroform	rat	liver,kidney	inh	chronic	LOEL	0.208	2.5E+01	ppm	3	5.18E+00			
EPA 1980c	chloroform	rat	reproduction	inh	chronic	LOEL	0.292	1.5E+02	mg/m3	30	8.58E-01			
Ecologistics 86b	chromium	chicken	growth	ingf	chronic	NOEL	1.000	5.0E+02	ppm	10	3.50E+00			CrIII
Ecologistics 86b	chromium	dairy cow		ingf	chronic	NOEL	1.000	3.0E+03	ppm	10	1.20E+01			CrIII
EPA 1980d	chromium	fish		aq	chronic	LOEL	1.000	1.0E+00	mg/L	3	3.40E-01	mg/L	fathead	CrVI
EPA 1980d	chromium	fish		aq	chronic	LOEL	1.000	2.0E+00	mg/L	3	6.67E-01	mg/L	fathead	CrIII
EPA 1980d	chromium	fish		aq	chronic	LOEL	1.000	7.3E-02	mg/L	3	2.43E-02	mg/L	trout	CrVI
Ecologistics 86b	chromium	mouse	lung	inh	chronic	LOEL	1.000	1.3E+01	mg/m3	3	7.28E+00			
NLM/HSDB	chromium	rat	liver	ingw	chronic	LOEL	1.000	1.3E+02	ppm	30	4.47E-01			CrVI
NRC 1980a	chromium	rat	LOEL	ingw	chronic	LOEL	1.000	2.5E+01	ppm	3	8.33E-01			CrVI
NRC 1980a	copper	calf	hepatic tox.	ingf	chronic	LOEL	1.000	1.2E+02	ppm	3	9.58E-01			

Documentation of Environmental Toxicity Data 3

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)		Tested Species	Notes
NRC 1977	copper	chicken	muscles	ingf	chronic	LOEL	1.000	1.0E+02	ppm	30	2.33E-01		--	
EPA 1984a	copper	fish		aq	chronic	LOEL	1.000	7.4E-03	mg/L	3	2.47E-03	mg/L	salmon	
EPA 1984a	copper	fish		aq	chronic	LOEL	1.000	6.0E-02	mg/L	3	2.00E-02	mg/L	pike	
EPA 1983a	copper	plant2	roots	soil	chronic	LOEL	1.000	6.9E+01	ppm	3	2.30E+01	ppm	corn	
Ecologistics 86b	copper	sheep	liver	ingf	chronic	LOEL	1.000	3.8E+01	ppm	3	3.62E-01			
NRC 1980	dichlorodifluoromethane	rat	none	gav	chronic	NOEL	1.000	1.5E+02	mg/kg/day	1	1.50E+02			
Spehar et al 79	ethanol	fish	LC50	aq	chronic	LOEL	1.000	1.1E+04	mg/L	500	2.24E+01	mg/L	trout	
Clayton 1982	ethanol	guinea pig	no effects	inh	chronic	NOEL	0.143	3.0E+03	ppm	10	4.94E+01			
Clayton 1982	ethanol	mouse	reproduction	ingw	chronic	LOEL	1.000	1.5E+05	ppm	30	1.00E+03			
Clayton 1982	ethanol	rat	wt., liver	ingw	chronic	LOEL	1.000	1.0E+04	mg/kg/day	3	3.40E+03			
Clayton 1982	ethanol	rat	reproduction	ingw	chronic	LOEL	1.000	1.5E+05	ppm	30	5.00E+02			
Crowell et al. 79	ethylene glycol	calf	death	gav	chronic	LOEL	1.000	2.2E+03	mg/kg/day	500	4.45E+00			
Union Carbide 85	ethylene glycol	rat	mineralization	ingf	chronic	NOEL	1.000	4.0E+01	mg/kg/day	1	4.00E+01			
USEPA 1985e	ethylene oxide	fish	LC50	aq	chronic	LOEL	1.000	8.4E+01	mg/L	500	1.68E-01	mg/L	minnow	
Snellings et al.84	ethylene oxide	rat	body wt.	inh	chronic	NOEL	0.179	1.0E+01	ppm	1	1.97E+00			
NRC 1981	formaldehyde	fish	LC50	aq	chronic	LOEL	1.000	3.7E+00	mg/L	500	7.40E-03	mg/L	bass	
NRC 1981	formaldehyde	plant1	injury	air	chronic	LOEL	0.208	7.0E-01	ppm	250	7.30E-04	mg/m3	alfalfa	
Ecologistics 85	formaldehyde	plant1	foliage	air	chronic	LOEL	1.000	2.0E-01	ppm	30	8.34E-03	mg/m3	petunia	
NRC 1981	formaldehyde	rat	weight	inh	chronic	NOEL	1.000	1.6E+00	ppm	1	1.20E+00			
NRC 1981	formaldehyde	rat	respiratory	inh	chronic	NOEL	0.179	4.0E+00	ppm	1	5.36E-01			
Ecologistics 86b	hexachlorobenzene	chicken	no effects	ingf	chronic	NOEL	1.000	1.0E+02	mg/kg/day	1	1.00E+02			
Hansen et al 78	hexachlorobenzene	chicken	egg production	gav	chronic	NOEL	1.000	5.0E+00	mg/kg/day	10	5.00E-01			
EPA 1985c	hexachlorobenzene	fish	no effects	aq	chronic	NOEL	1.000	2.6E-02	mg/L	1	2.60E-02	mg/L	bass	
EPA 1985c	hexachlorobenzene	mink	reproduction	ingf	chronic	LOEL	1.000	1.0E+00	ppm	10	1.50E-02			
EPA 1985c	hexachlorobenzene	mouse	growth	ingf	chronic	LOEL	1.000	6.0E+00	mg/kg/day	3	2.00E+00			
Ecologistics 86a	hexachlorobenzene	plant2	no effects	soil	chronic	NOEL	1.000	2.0E+00	ppm	1	2.00E+00	ppm	lettuce	
EPA 1985c	hexachlorobenzene	quail	egg production	ingf	chronic	NOEL	1.000	1.0E+00	mg/kg/day	1	1.00E+00			
EPA 1985c	hexachlorobenzene	rat	immune	ingf	chronic	LOEL	1.000	5.0E+01	ppm	3	1.00E+00			
EPA 1985c	hexachlorobenzene	rat	liver	ingf	chronic	NOEL	1.000	5.0E-01	mg/kg/day	1	5.00E-01			
Ecologistics 86b	hexachlorobenzene	sheep	growth	ingf	chronic	NOEL	1.000	4.3E+00	mg/kg/day	1	4.30E+00			
BLM/HSDB	hydrazine	fish	larval effects	aq	chronic	NOEL	1.000	1.0E-01	mg/L	1	1.00E-01	mg/L	trout	
ACGIH 1986	hydrazine	mouse	liver	inh	chronic	LOEL	1.000	2.0E-01	ppm	2.5	1.79E-01			
Clayton 1982	hydrazine	rat	death	inh	chronic	LOEL	0.250	5.0E+00	ppm	2.5	4.01E-01			
EPA 1985a	hydrogen chloride	fish	LC50 or EC50	aq	acute	LOEL	1.000	4.0E-02	mg/L	5	8.00E-03	mg/L	shiner	
EPA 1985a	hydrogen chloride	fish	reproduction	aq	chronic	NOEL	1.000	1.1E-02	mg/L	1	1.12E-02	mg/L	minnow	

Documentation of Environmental Toxicity Data 4

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)		Tested Species	Notes
NRC 1976	hydrogen chloride	guinea pig	irritation	inh	chronic	LOEL	0.250	1.5E+02	mg/m3	30	7.50E-01		--	
Ecologistics 86b	hydrogen chloride	mouse	respiratory	inh	acute	LOEL	0.207	2.5E+01	mg/m3	2.5	1.18E-01			
Ecologistics 86b	hydrogen chloride	mouse	LC50	inh	acute	LOEL	0.021	3.9E+03	mg/m3	5	2.76E+01			
Ecologistics 85	hydrogen chloride	plant1	chronic effects	air	chronic	LOEL	1.000	6.0E-01	ppm	3	2.03E-01	mg/m3	plant	
Ecologistics 85	hydrogen chloride	plant1	foliage	air	acute	LOEL	0.083	6.0E+00	ppm	2.5	2.03E-01	mg/m3	tomato	
Ecologistics 85	hydrogen chloride	plant1	injury threshold	air	acute	NOEL	0.042	6.6E-01	ppm	1	2.91E-02	mg/m3	buckwheat	
Ecologistics 85	hydrogen chloride	plant1	acute effects	air	acute	LOEL	1.000	1.0E+00	ppm	2.5	6.08E-01	mg/m3	plant	
NRC 1976	hydrogen chloride	rabbit	irritation	inh	chronic	LOEL	0.250	1.5E+02	mg/m3	30	9.38E-01			
EPA 1980a	hydrogen cyanide	mouse	respiration	inh	acute	LOEL	1.000	1.0E+01	mg/m3	250	6.72E-02			
EPA 1981a	hydrogen cyanide	rat	no effect	ingw	chronic	NOEL	1.000	1.9E-01	mg/L	1	1.90E-02			
Ecologistics 86b	hydrogen fluoride	calf	death	gav	acute	LOEL	1.000	4.5E+01	mg/kg/day	5	9.04E+00			
Ecologistics 86b	hydrogen fluoride	chicken	egg production	ingf	chronic	LOEL	1.000	3.5E+02	ppm	30	8.17E-01			
NRCC 1977	hydrogen fluoride	fish	death	aq	chronic	LOEL	1.000	5.0E+00	mg/L	100	5.00E-02	mg/L	trout	
Ecologistics 86b	hydrogen fluoride	goat	death	gav	acute	LOEL	1.000	1.0E+02	mg/kg/day	5	1.99E+01			
Clayton 1981	hydrogen fluoride	guinea pig	tolerable	inh	acute	NOEL	1.000	1.5E+01	mg/m3	1	9.00E+00			
NRCC 1977	hydrogen fluoride	plant1	vigor	air	chronic	LOEL	1.000	2.1E-03	mg/m3	3	7.00E-04	mg/m3	beans	
Ecologistics 85	hydrogen fluoride	plant1	OMOE standard	air	acute	NOEL	0.021	6.3E-03	mg/m3	1	8.96E-05	mg/m3	plants	
Ecologistics 85	hydrogen fluoride	plant1	foliar necrosis	air	chronic	LOEL	1.000	1.7E-04	mg/m3	3	5.67E-05	mg/m3	grape	
Ecologistics 86a	hydrogen fluoride	plant2	phytotoxicity	soil	chronic	LOEL	1.000	2.0E+02	ppm	3	6.67E+01	ppm	grape	
NRCC 1977	hydrogen fluoride	quail	bone strength	ingf	chronic	LOEL	1.000	7.5E+02	ppm	3	2.21E+01			
Clayton 1981	hydrogen fluoride	rabbit	weight	inh	chronic	LOEL	0.250	7.0E+00	mg/m3	30	4.38E-02			
Clayton 1981	hydrogen fluoride	rabbit	tolerable	inh	acute	NOEL	1.000	1.5E+01	mg/m3	1	1.13E+01			
Ecologistics 86b	hydrogen fluoride	steer	NRC max. tol.	ingw	chronic	NOEL	1.000	2.0E+00	mg/L	1	1.60E-01			
Ecologistics 86b	hydrogen fluoride	steer	Ontario standard	ingf	chronic	NOEL	1.000	3.5E+01	mg/kg/day	1	3.50E+01			
Ecologistics 86b	hydrogen fluoride	steer	food consumption	ingf	acute	LOEL	1.000	6.0E+02	ppm	2.5	5.76E+00			
Ecologistics 86b	hydrogen fluoride	steer	max. permissible	inh	chronic	NOEL	1.000	8.0E-02	mg/m3	1	2.00E-02			
Ecologistics 86b	hydrogen sulfide	calf	irritation	inh	chronic	LOEL	1.000	2.8E+01	mg/m3	30	1.40E-01			
Ecologistics 86b	hydrogen sulfide	chicken	respiratory	inh	acute	NOEL	0.021	7.5E+02	mg/m3	1	1.56E+01			
Ecologistics 86b	hydrogen sulfide	dairy cow	irritation	inh	chronic	LOEL	1.000	2.8E+01	mg/m3	30	2.59E-01			
NRC 1979	hydrogen sulfide	fish	death	aq	chronic	NOEL	1.000	2.0E-03	mg/L	1	2.00E-03	mg/L	G. pseudoli.	
Verschueren 1983	hydrogen sulfide	mouse	LC50	inh	acute	LOEL	0.013	1.5E+03	mg/m3	5	6.30E+00			
Ecologistics 86b	hydrogen sulfide	pig	not reported	inh	acute	NOEL	0.083	7.0E+01	mg/m3	1	6.67E-01			
Ecologistics 85	hydrogen sulfide	plant1	injury	air	acute	LOEL	0.188	2.8E+01	mg/m3	2.5	2.10E+00	mg/m3	tomato	
NRC 1979	hydrogen sulfide	plant1	growth	air	chronic	NOEL	1.000	3.0E-02	ppm	1	6.26E-02	mg/m3	alfalfa,grape	
Ecologistics 86b	hydrogen sulfide	rabbit	death	inh	acute	LOEL	1.000	1.4E+03	mg/m3	5	2.10E+02			

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)	Tested Species	Notes
Verschueren 1983	hydrogen sulfide	rat	LC50	inh	acute	LOEL	0.010	1.5E+03	mg/m3	5	1.75E+00		
Ecologistics 86b	hydrogen sulfide	steer	max. acceptable	inh	chronic	NOEL	1.000	2.7E-01	mg/m3	1	6.75E-02		
Demayo et al. 1982	lead	calf	toxicity	ingf	chronic	LOEL	1.000	5.0E+00	mg/kg/day	30	1.67E-01		
Ecologistics 86b	lead	dairy cow	gen. tox.	ingf	chronic	LOEL	0.429	9.0E+00	mg/kg/day	30	1.29E-01		
Ecologistics 86b	lead	duck		ingf	chronic	NOEL	1.000	6.0E+00	mg/kg/day	1	6.00E+00		
Demayo et al. 1982	lead	fish		aq	chronic	LOEL	1.000	7.6E-03	mg/L	3	2.53E-03 mg/L		
EPA 1980r	lead	fish		aq	chronic	LOEL	1.000	9.2E-02	mg/L	3	3.07E-02 mg/L	bluegill	methyl Hg
Demayo et al. 1982	lead	plant2	roots	soil	chronic	LOEL	1.000	2.1E+02	ppm	3	6.99E+01 ppm	pine	methyl Hg
IARC 80r	lead	mouse	fetal death	ingw	chronic	LOEL	1.000	5.0E+00	mg/kg/day	30	1.67E-01		methyl Hg
Ecologistics 86b	mercury	calf	none	ingf	chronic	NOEL	1.000	1.0E-01	mg/kg/day	10	1.00E-02		
Ecologistics 86b	mercury	duck	none	ingf	chronic	NOEL	1.000	3.5E+00	ppm	10	3.50E-02		
EPA 1980g	mercury	fish	toxicity	aq	chronic	LOEL	1.000	5.2E-06	mg/L	3	1.73E-06 mg/L	trout	
EPA 1980g	mercury	mouse	reproduction	gav	chronic	LOEL	1.000	2.6E+00	mg/kg/day	30	8.73E-02		
NLM/HSDB	methyl ethyl ketone	fish	TLm	aq	chronic	LOEL	1.000	5.6E+03	mg/L	500	1.12E+01 mg/L	mosquito fish	
Clayton 1982	methyl ethyl ketone	rat	no effects	inh	chronic	NOEL	1.000	1.1E+03	ppm	1	2.05E+03		
Clayton 1982	methyl ethyl ketone	rat	reproduction	inh	chronic	LOEL	0.292	1.0E+03	ppm	30	1.75E+01		
EPA 1985d	methylene chloride	fish	sublethal	aq	acute	LOEL	1.000	9.9E+01	mg/L	2.5	3.96E+01 mg/L	minnow	
EPA 1985d	methylene chloride	fish	LC50	aq	acute	LOEL	1.000	1.9E+02	mg/L	5	3.86E+01 mg/L	minnow	
EPA 1985d	methylene chloride	fish	survival	aq	chronic	LOEL	1.000	4.1E-01	mg/L	10	4.10E-02 mg/L	trout	
EPA 1985d	methylene chloride	guinea pig	LC50	inh	acute	LOEL	0.250	1.2E+04	ppm	5	1.22E+03		
EPA 1985d	methylene chloride	mouse	decr. activity	inh	acute	LOEL	0.125	1.0E+03	ppm	2.5	2.97E+02		
EPA 1985d	methylene chloride	mouse	behavior	inh	chronic	NOEL	1.000	1.0E+02	ppm	1	5.95E+02		
EPA 1985d	methylene chloride	mouse	LD50	gav	acute	LOEL	1.000	2.0E+03	mg/kg/day	5	3.97E+02		
EPA 1985d	methylene chloride	plant1	red photosyn	air	chronic	LOEL	1.000	2.1E+05	ppm	250	2.97E+03 mg/m3	alfalfa	
EPA 1985d	methylene chloride	plant1	red. photosyn.	air	acute	LOEL	1.000	2.1E+05	ppm	2.5	2.97E+05 mg/m3	alfalfa	
EPA 1985d	methylene chloride	rat	CNS effects	inh	acute	LOEL	0.063	2.6E+04	ppm	2.5	1.36E+03		
EPA 1985d	methylene chloride	rat	kidney	inh	chronic	NOEL	1.000	2.5E+01	ppm	1	5.31E+01		
EPA 1980h	naphthalene	fish	tox.	aq	chronic	LOEL	1.000	6.2E-01	mg/L	3	2.07E-01 mg/L	fathead	
Millemann et al.84	naphthalene	fish	LC50	aq	chronic	LOEL	1.000	1.2E-01	mg/L	100	1.20E-03 mg/L	trout	
Shopp 1984	naphthalene	mouse	none	gav	chronic	NOEL	1.000	5.3E+01	mg/kg/day	10	5.30E+00		
EPA 1980h	naphthalene	rat	cataracts	ingf	chronic	LOEL	1.000	2.0E+04	ppm	30	4.00E+01		
Ecologistics 86b	nickel	calf		ingf	chronic	NOEL	1.000	6.3E+01	ppm	10	1.58E-01		
Ecologistics 86b	nickel	chicken	growth	ingf	chronic	NOEL	1.000	3.0E+02	ppm	10	2.19E+00		
EPA 1980i	nickel	fish		aq	chronic	LOEL	1.000	1.1E-01	mg/L	3	3.63E-02 mg/L	fathead	
EPA 1980i	nickel	fish		aq	chronic	LOEL	1.000	3.5E-01	mg/L	3	1.17E-01 mg/L	trout	H02

Documentation of Environmental Toxicity Data 6

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)		Tested Species	Notes
EPA 1980a	nickel	plant2	yield	soil	chronic	LOEL	1.000	8.0E+01	ppm	3	2.67E+01	ppm	lettuce	NO2
EPA 1980b	nickel	rat	reproduction	ingw	chronic	LOEL	1.000	5.0E+00	ppm	3	1.67E-01			NO2
NRC 1977	nitrogen oxides	guinea pig	respiration	inh	acute	LOEL	1.000	1.2E+02	mg/m3	250	2.93E-01			NO2
NRC 1977	nitrogen oxides	mouse	lung	inh	chronic	LOEL	1.000	6.0E-01	mg/m3	3	3.36E-01			NO2
NRC 1977	nitrogen oxides	mouse	immune system	inh	acute	LOEL	1.000	6.6E+00	mg/m3	500	2.22E-02			NO2
Ashenden 1979	nitrogen oxides	plant1	yield	air	chronic	LOEL	1.000	1.9E-01	mg/m3	3	6.47E-02	mg/m3	plant	
NRC 1977	nitrogen oxides	plant1	growth	air	chronic	LOEL	1.000	4.7E-01	mg/m3	3	1.57E-01	mg/m3	tomato	analogy to p-NP
NRC 1977	nitrogen oxides	rat	tachypnea	inh	chronic	LOEL	1.000	1.5E+00	mg/m3	3	3.00E-01			
EPA 1980j	o-nitrophenol	fish	LC50	aq	chronic	LOEL	1.000	8.3E+00	mg/L	500	1.66E-02	mg/L	bluegill	
NRC 1982	o-nitrophenol	guinea pig	cataracts	inj	chronic	LOEL	1.000	8.3E+00	mg/kg/day	30	2.77E-01			
Ecologistics 86b	PCBs	chicken	laying	ingf	chronic	LOEL	1.000	1.0E+01	ppm	3	2.33E-01			
EPA 1980l	PCBs	fish		aq	chronic	LOEL	1.000	2.0E-04	mg/L	3	6.67E-05	mg/L	fathead	
EPA 1980l	PCBs	fish		aq	chronic	LOEL	1.000	1.0E-03	mg/L	3	3.33E-04	mg/L	trout	
EPA 1984f	PCBs	mink	reproduction	ingf	chronic	LOEL	1.000	2.0E+00	mg/kg/day	3	6.67E-01			
EPA 1984f	PCBs	mouse	hepatic	ingf	chronic	LOEL	1.000	3.8E+00	mg/kg/day	3	1.25E+00			TCDD
Ecologistics 86b	PCBs	pig	reproduction	ingf	chronic	LOEL	1.000	2.0E+01	ppm	3	1.67E-01			TCDD
Ecologistics 86a	PCBs	plant2	growth	soil	chronic	LOEL	1.000	1.0E+02	ppm	3	3.33E+01	ppm	soybean	TCDD
EPA 1984b	PCDD	fish	lethal	aq	chronic	LOEL	1.000	2.4E-06	mg/L	100	2.40E-08	mg/L	catfish	based on naphtha
EPA 1984b	PCDD	fish	growth	aq	chronic	LOEL	1.000	1.0E-06	mg/L	3	3.33E-07	mg/L	trout	based on naphtha
Kociba 1979	PCDD	rat	hepatic	ingf	chronic	NOEL	1.000	1.0E-06	mg/kg/day	1	1.00E-06			
Verschueren 1983	petroleum distillates	fish	LC50	aq	chronic	LOEL	1.000	4.2E+00	mg/L	500	8.40E-03	mg/L	bluegill	
Carpenter et al 75	petroleum distillates	rat	no effects	inh	chronic	NOEL	0.179	1.2E+03	ppm	1	6.96E+02			
DeGraeve et al. 80	phenol	fish	growth	aq	chronic	LOEL	1.000	2.0E-01	mg/L	3	6.67E-02	mg/L	trout	
Ecologistics 86b	phenol	plant2	acute tox.	soil	chronic	LOEL	1.000	1.0E+02	ppm	250	4.00E-01	ppm	plant	
EPA 1980k	phenol	rat	kidney	gav	chronic	LOEL	0.750	5.0E+01	mg/kg/day	3	1.25E+01			
EPA 1980k	phenol	rat	reproduction	inh	chronic	LOEL	0.208	5.0E-01	mg/m3	30	2.08E-03			
Fein-Marquart	pyridine	fish	LC50	aq	chronic	LOEL	1.000	2.6E+01	mg/L	500	5.20E-02	mg/L	carp	
EOSH 1971	pyridine	rat	liver	inh	chronic	LOEL	0.208	1.0E+01	ppm	3	1.37E+00			
Fein-Marquart	pyridine	fish	LC50	aq	acute	LOEL	1.000	2.6E+01	mg/L	5	5.20E+00	mg/L	carp	
Fein-Marquart	styrene	fish	LC50	aq	chronic	LOEL	1.000	2.2E+01	mg/L	500	4.40E-02	mg/L	bluegill	
Fein-Marquart	styrene	fish	LC50	aq	chronic	LOEL	1.000	1.7E+01	mg/L	500	3.40E-02	mg/L	Leuciscus l.	
NIOSH 1983	styrene	guinea pig	lung tox.	inh	chronic	NOEL	1.000	6.5E+02	ppm	1	1.69E+03			
Ecolog stics 86b	styrene	mouse	LD50	gav	chronic	LOEL	1.000	3.2E+02	mg/kg/day	250	1.26E+00			
NIOSH 1983	styrene	rat	reproduction	inh	chronic	NOEL	1.000	1.2E+00	ppm	10	3.12E-01			
EPA 1982	sulfur dioxide	guinea pig	lung	inh	chronic	NOEL	1.000	9.0E-01	mg/m3	1	5.40E-01			

Citation	Chemical	Species	End Point	Exposure Route(1)	Acute or Chronic?(2)	Data Measure	Adjustment Factor(3)	Exposure	Units	Safety Factor(3)	Safe Systemic Dose (mg/kg/day unless shown)	Tested Species	Notes
Ecologistics 86b	sulfur dioxide	guinea pig	lung	inh	chronic	LOEL	1.000	6.0E-02	ppm	30	3.20E-03	--	
EPA 1982	sulfur dioxide	mouse	LC50	inh	acute	LOEL	1.000	6.1E+02	ppm	250	1.09E+01		
Ashenden 1979	sulfur dioxide	plant1	leaves	air	chronic	LOEL	1.000	1.9E-01	mg/m3	3	6.47E-02 mg/m3	grass	
EPA 1980n	tetrachloroethylene	fish	tox.	aq	chronic	LOEL	1.000	8.4E-01	mg/L	3	2.80E-01 mg/L	fathead	
EPA 1985f	tetrachloroethylene	guinea pig	liver	inh	chronic	LOEL	0.208	6.8E+02	mg/m3	3	2.83E+01		
EPA 1985f	tetrachloroethylene	rabbit	liver	inh	chronic	LOEL	0.146	1.5E+01	ppm	3	3.78E+00		
Fein-Marquart	tetraethyl lead	fish	lethal	aq	chronic	LOEL	1.000	2.0E-01	mg/L	500	4.00E-04 mg/L	bluegill	
Blakley et al.1980	tetraethyl lead	mouse	immune tox.	ingw	chronic	LOEL	1.000	5.0E-01	ppm	30	3.33E-03		
ACGIH 1980	tetraethyl lead	rat	histopath.	ingf	chronic	LOEL	1.000	1.7E-03	mg/kg/day	3	5.67E-04		
NLM/HSDB	toluene	fish	LC50	aq	chronic	LOEL	1.000	1.3E+01	mg/L	500	2.60E-02 mg/L	trout	
EPA 1980o	toluene	fish		aq	chronic	LOEL	1.000	5.0E+00	mg/L	3	1.67E+00 mg/L	sheepshead(s.w.)	
NLM/HSDB	toluene	mouse	teratogenicity	gav	chronic	LOEL	1.000	1.0E+00	mg/kg/day	30	3.33E-02		
EPA 1982	toluene	rat	blood	inh	chronic	NOEL	1.000	3.0E+01	ppm	1	6.91E+01		
EPA 1980b	trichloroethane	fish	tox.	aq	chronic	LOEL	1.000	9.4E+00	mg/L	3	3.13E+00 mg/L	minnow	
EPA 1984c	trichloroethane	mouse	liver	inh	chronic	LOEL	1.000	2.5E+02	ppm	30	7.78E+01		
EPA 1984c	trichloroethane	rabbit	weight	inh	chronic	LOEL	1.000	3.7E+02	ppm	3	5.14E+02		
NRC 1981	trichloroethane	rat	weight	gav	chronic	LOEL	0.714	7.5E+02	mg/kg/day	3	1.79E+02		
EPA 1980p	trichloroethylene	fish	equilibrium	aq	chronic	LOEL	1.000	2.2E+01	mg/L	250	8.76E-02 mg/L	fathead	
EPA 1985g	trichloroethylene	mouse	liver	gav	chronic	LOEL	1.000	2.5E+02	mg/kg/day	30	8.33E+00		
NRC 1980	trichloroethylene	rat	liver	inh	chronic	LOEL	0.238	5.5E+01	ppm	3	1.43E+01		
EPA 1985g	trichloroethylene	rat	immunity	ingw	chronic	LOEL	1.000	1.0E+02	mg/L	3	3.33E+00		
ACGIH 1986	trichlorotrifluoroethane	rat	none	inh	chronic	NOEL	0.083	1.2E+04	ppm	1	4.68E+03		
NRC 1981a	xylene	fish	LC50	aq	chronic	LOEL	1.000	1.4E+01	mg/L	500	2.70E-02 mg/L	trout	
Ecologistics 86b	xylene	mouse	embryotox.	inh	chronic	NOEL	1.000	1.0E+01	mg/m3	10	1.68E+00		
Ecologistics 86b	xylene	rat		inh	chronic	NOEL	1.000	7.8E+01	ppm	10	2.07E+01		
EPA 1980q	zinc	fish		aq	chronic	NOEL	1.000	5.3E-01	mg/L	1	5.34E-01 mg/L	trout	
EPA 1980q	zinc	fish		aq	chronic	LOEL	1.000	1.0E-01	mg/L	3	3.33E-02 mg/L	fathead	
EPA 1980q	zinc	mouse		ingw	chronic	LOEL	1.000	1.0E+02	mg/kg/day	3	3.33E+01		
Kabata-Pendias 84	zinc	plant2	toxicity	soil	chronic	LOEL	1.000	7.0E+01	ppm	3	2.33E+01 ppm	plants	
NRC 1980a	zinc	quail	blood	ingf	chronic	NOEL	1.000	1.3E+02	ppm	10	1.10E+00		
Ecologistics 86b	zinc	steer	growth	ingf	chronic	NOEL	1.000	1.0E+02	ppm	3	8.00E-01		

Documentation of Environmental Toxicity Data: Footnotes to Table L-1

(1) air = ambient air; aq = aquatic (ambient water); gav = gavage; ingf = ingestion via feed; ingw = ingestion via drinking water; inh = inhalation; inj = injection; soil = soil (plants).

(2) "Acute or chronic?" refers to the type of exposure associated with the safe systemic dose and not necessarily to the nature of the experimental exposure.

(3) Adjustment Factors

Adjustment factors are used to adjust the experimental data for less than continuous (or daily) exposure so that criterion doses can be directly compared to estimated systemic doses. For example, the adjustment factor for an inhalation toxicity study involving exposures of 6 hours/day, 5 days/week would be as follows:

$$\frac{6\ \text{hours/day}}{24\ \text{hours/day}} \times \frac{5\ \text{days/week}}{7\ \text{days/week}} = 0.18$$

Similarly, for a gavage study involving dosing for 5 days/week, the adjustment factor would be calculated as follows:

$$\frac{5\ \text{days/week}}{7\ \text{days/week}} = 0.71$$

Safety Factors

Refer to Appendix K for a discussion of safety factors applied in the environmental risk assessment.

Note regarding plant toxicity data:

Plant1 indicates data from a toxicity study involving exposure of plants to airborne substances.

Plant2 indicates data from a toxicity study involving exposure of plants to substances in the soil.

TABLE L-2

CRITERION DOSES AND CONCENTRATIONS FOR CHRONIC EXPOSURE

Note:
Criterion doses in mg/kg/d,
Criterion concentrations for fish in mg/L
Criterion concentrations for plant 1 in mg/m^3
Criterion concentrations for plant 2 in ppm

TABLE L-2
Criterion Doses

CRITERION DOSES 8/10/87

Chemical Name	calf	chicken	dairy cow	duck	fish	goat	guinea pig
acetone	NA	NA	1.15E+01 *	NA	7.90E-01	NA	NA
acrylonitrile	NA	NA	6.58E-02 *	NA	2.20E-02	NA	NA
arsenic	NA	1.17E-03	4.68E-01	NA	1.00E-02	NA	NA
benzene	NA	NA	NA	NA	1.06E-03	NA	NA
benzo(a)anthracene	NA	NA	6.67E-02 *	NA	2.00E-04	NA	NA
benzo(a)pyrene	NA	NA	3.33E-02 *	NA	NA	NA	NA
beryllium	NA	NA	6.80E-04 *	NA	2.60E-04	NA	NA
bromodichloromethane	NA	NA	2.00E+00 *	NA	NA	NA	NA
bromoform	NA	NA	3.00E-01 *	NA	5.86E-03	NA	NA
butadiene	NA	NA	2.25E-01 *	NA	1.43E-02	NA	NA
cadmium	NA	2.10E-02	6.67E-02	1.50E-01	6.67E-05	NA	NA
carbon tetrachloride	NA	NA	3.66E-01 *	NA	3.40E-01	NA	3.66E-01
chlorobenzene	NA	NA	7.14E-01 *	NA	7.00E-02	NA	NA
chloroform	NA	NA	8.58E-02 *	NA	1.24E-02	NA	NA
chromium	NA	3.50E-01	1.20E+00	NA	2.43E-03	NA	NA
copper	9.58E-02	2.33E-02	NA	NA	2.47E-04	NA	NA
dichlorodifluoromethane	NA	NA	1.50E+01 *	NA	NA	NA	NA
ethanol	NA	NA	4.94E+00 *	NA	2.24E+00	NA	4.94E+00
ethylene glycol	4.45E-01	NA	NA	NA	NA	NA	NA
ethylene oxide	NA	NA	1.97E-01 *	NA	1.68E-02	NA	NA
formaldehyde	NA	NA	5.36E-02 *	NA	7.40E-04	NA	NA
hexachlorobenzene	NA	5.00E-02	NA	NA	2.60E-03	NA	NA
hydrazine	NA	NA	1.79E-02 *	NA	1.00E-02	NA	NA
hydrogen chloride	NA	NA	7.50E-02 *	NA	1.12E-03	NA	7.50E-02
hydrogen cyanide	NA	NA	1.90E-03 *	NA	NA	NA	NA
hydrogen fluoride	NA	8.17E-02	NA	NA	NA	NA	NA
hydrogen sulfide	1.40E-02	NA	2.59E-02	NA	2.00E-04	NA	NA
lead	1.67E-02	NA	1.29E-02	6.00E-01	2.53E-04	NA	NA
mercury	1.00E-03	NA	NA	3.30E-03	1.73E-05	NA	NA
methyl ethyl ketone	NA	NA	1.75E+00 *	NA	1.12E+00	NA	NA
methylene chloride	NA	NA	5.31E+00 *	NA	4.10E-03	NA	NA
naphthalene	NA	NA	5.30E-01 *	NA	1.20E-04	NA	NA
nickel	1.58E-02	2.10E-01	NA	NA	3.63E-03	NA	NA
nitrogen oxides	NA	NA	3.00E-02 *	NA	NA	NA	NA
o-nitrophenol	NA	NA	2.77E-02 *	NA	1.66E-03	NA	2.77E-02
PCBs	NA	2.33E-02	NA	NA	6.67E-06	NA	NA
PCDD	NA	NA	1.00E-07 *	NA	2.40E-09	NA	NA
petroleum distillates	NA	NA	6.96E+01 *	NA	8.40E-04	NA	NA
phenol	NA	NA	2.08E-04 *	NA	6.67E-03	NA	NA
pyridine	NA	NA	1.37E-01 *	NA	5.20E-03	NA	NA
styrene	NA	NA	3.12E-02 *	NA	3.40E-03	NA	1.69E+02
sulfur dioxide	NA	NA	3.20E-04 *	NA	NA	NA	3.20E-04
tetrachloroethylene	NA	NA	3.78E-01 *	NA	2.80E-02	NA	2.82E+00
tetraethyl lead	NA	NA	4.05E-05 *	NA	4.00E-05	NA	NA
toluene	NA	NA	3.33E-03 *	NA	2.60E-03	NA	NA
trichloroethane	NA	NA	1.79E+01 *	NA	3.13E-01	NA	NA
trichluroethylene	NA	NA	3.33E-01 *	NA	8.76E-03	NA	NA
trichlorotrifluoroethan	NA	NA	4.68E+02 *	NA	NA	NA	NA
xylene	NA	NA	1.68E-01 *	NA	2.70E-03	NA	NA
zinc	NA	NA	NA	NA	3.33E-03	NA	NA

CRITERION DOSES

Chemical Name	heron	horse	mink	mouse	pig	plant1	plant2
acetone	NA	NA	NA	NA	NA	NA	NA
acrylonitrile	NA	NA	NA	NA	NA	NA	NA
arsenic	1.17E-03 *	5.30E-02	NA	NA	NA	NA	7.33E-01
benzene	NA	NA	NA	1.00E-03	9.30E-03	1.20E-02	NA
benzo(a)anthracene	NA	NA	NA	NA	NA	NA	NA
benzo(a)pyrene	NA	NA	NA	3.33E-02	NA	NA	NA
beryllium	NA	NA	NA	NA	NA	NA	3.33E-02
bromodichloromethane	NA	NA	NA	2.00E+00	NA	NA	NA
bromoform	NA	NA	NA	4.17E+00	NA	NA	NA
butadiene	NA	NA	NA	NA	NA	NA	NA
cadmium	2.10E-02 *	NA	NA	NA	NA	NA	8.33E-02
carbon tetrachloride	NA	NA	NA	NA	NA	NA	NA
chlorobenzene	NA	NA	NA	7.14E-01	NA	NA	NA
chloroform	NA	NA	NA	1.71E+00	NA	NA	NA
chromium	3.50E-01 *	NA	NA	7.28E-01	NA	NA	NA
copper	2.33E-02 *	NA	NA	NA	NA	NA	2.30E+00
dichlorodifluoromethane	NA	NA	NA	NA	NA	NA	NA
ethanol	NA	NA	NA	1.00E+02	NA	NA	NA
ethylene glycol	NA	NA	NA	NA	NA	NA	NA
ethylene oxide	NA	NA	NA	NA	NA	NA	NA
formaldehyde	NA	NA	NA	NA	NA	7.30E-05	NA
hexachlorobenzene	5.00E-02 *	NA	1.50E-03	2.00E-01	NA	NA	2.00E-01
hydrazine	NA	NA	NA	1.79E-02	NA	NA	NA
hydrogen chloride	NA	NA	NA	NA	NA	2.03E-02	NA
hydrogen cyanide	NA	NA	NA	NA	NA	NA	NA
hydrogen fluoride	8.17E-02 *	NA	NA	NA	NA	5.67E-06	6.67E+00
hydrogen sulfide	NA	NA	NA	NA	NA	4.26E-03	NA
lead	6.00E-01 *	NA	NA	1.67E-02	NA	NA	6.90E+00
mercury	3.30E-03 *	NA	NA	8.33E-03	NA	NA	NA
methyl ethyl ketone	NA	NA	NA	NA	NA	NA	NA
methylene chloride	NA	NA	NA	5.95E+01	NA	2.97E+02	NA
naphthalene	NA	NA	NA	5.30E-01	NA	NA	NA
nickel	2.10E-01 *	NA	NA	NA	NA	NA	2.67E+00
nitrogen oxides	NA	NA	NA	3.36E-02	NA	6.47E-03	NA
o-nitrophenol	NA	NA	NA	NA	NA	NA	NA
PCBs	2.33E-02 *	NA	6.67E-02	1.25E-01	1.67E-02	NA	3.33E+00
PCDD	NA	NA	NA	NA	NA	NA	NA
petroleum distillates	NA	NA	NA	NA	NA	NA	NA
phenol	NA	NA	NA	NA	NA	NA	4.00E-02
pyridine	NA	NA	NA	NA	NA	NA	NA
styrene	NA	NA	NA	1.26E-01	NA	NA	NA
sulfur dioxide	NA	NA	NA	NA	NA	6.47E-03	NA
tetrachloroethylene	NA	NA	NA	NA	NA	NA	NA
tetraethyl lead	NA	NA	NA	3.33E-04	NA	NA	NA
toluene	NA	NA	NA	3.33E-03	NA	NA	NA
trichloroethane	NA	NA	NA	7.78E+00	NA	NA	NA
trichloroethylene	NA	NA	NA	8.33E-01	NA	NA	NA
trichlorotrifluoroethan	NA	NA	NA	NA	NA	NA	NA
xylene	NA	NA	NA	1.68E-01	NA	NA	NA
zinc	3.68E-02 *	NA	NA	3.33E+00	NA	NA	2.33E+00

CRITERION DOSES

Chemical Name	quail	rabbit	rat	sheep	steer
acetone	NA	NA	1.15E+01	NA	NA
acrylonitrile	NA	6.58E-02	1.17E-01	NA	NA
arsenic	NA	NA	4.17E-01	4.89E-02	NA
benzene	NA	3.33E-02	7.10E-02	NA	NA
benzo(a)anthracene	NA	NA	6.67E-02	NA	NA
benzo(a)pyrene	NA	NA	NA	NA	NA
beryllium	NA	NA	6.80E-04	NA	NA
bromodichloromethane	NA	NA	NA	NA	NA
bromoform	NA	NA	3.00E-01	NA	NA
butadiene	NA	NA	2.25E-01	NA	NA
cadmium	NA	NA	7.50E-02	NA	NA
carbon tetrachloride	NA	NA	3.66E-01	NA	NA
chlorobenzene	NA	NA	4.40E+00	NA	NA
chloroform	NA	NA	8.58E-02	NA	NA
chromium	NA	NA	4.47E-02	NA	NA
copper	NA	NA	NA	3.62E-02	NA
dichlorodifluoromethane	NA	NA	1.50E+01	NA	NA
ethanol	NA	NA	5.00E+01	NA	NA
ethylene glycol	NA	NA	4.00E+00	NA	NA
ethylene oxide	NA	NA	1.97E-01	NA	NA
formaldehyde	NA	NA	5.36E-02	NA	NA
hexachlorobenzene	1.00E-01	NA	5.00E-02	4.30E-01	NA
hydrazine	NA	NA	4.01E-02	NA	NA
hydrogen chloride	NA	9.37E-02	NA	NA	NA
hydrogen cyanide	NA	NA	1.90E-03	NA	NA
hydrogen fluoride	2.21E+00	4.37E-03	NA	NA	2.00E-03
hydrogen sulfide	NA	NA	NA	NA	6.75E-03
lead	NA	NA	NA	NA	NA
mercury	NA	NA	NA	NA	NA
methyl ethyl ketone	NA	NA	1.75E+00	NA	NA
methylene chloride	NA	NA	5.31E+00	NA	NA
naphthalene	NA	NA	4.00E+00	NA	NA
nickel	NA	NA	1.67E-02	NA	NA
nitrogen oxides	NA	NA	3.00E-02	NA	NA
o-nitrophenol	NA	NA	NA	NA	NA
PCBs	NA	NA	NA	NA	NA
PCDD	NA	NA	1.00E-07	NA	NA
petroleum distillates	NA	NA	6.96E+01	NA	NA
phenol	NA	NA	2.08E-04	NA	NA
pyridine	NA	NA	1.37E-01	NA	NA
styrene	NA	NA	3.12E-02	NA	NA
sulfur dioxide	NA	NA	NA	NA	NA
tetrachloroethylene	NA	3.78E-01	NA	NA	NA
tetraethyl lead	NA	NA	5.67E-05	NA	NA
toluene	NA	NA	6.91E+00	NA	NA
trichloroethane	NA	5.14E+01	1.79E+01	NA	NA
trichloroethylene	NA	NA	3.33E-01	NA	NA
trichlorotrifluoroethan	NA	NA	4.68E+02	NA	NA
xylene	NA	NA	2.07E+00	NA	NA
zinc	1.10E-01	NA	NA	NA	8.00E-02

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APPENDIX M

ANALYSIS OF CONTAMINANT RELEASES FROM THE EVAPORATOR STACK AND THE STABILIZED LANDFILL

(As Conducted by the Engineering Consultants to OWMC)

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APPENDIX M

ANALYSIS OF CONTAMINANT RELEASES FROM THE EVAPORATOR STACK AND THE STABILIZED LANDFILL

(As Conducted by the Engineering Consultants to OWMC)

The proposed OWMC Facility will include a physical/chemical plant consisting of two separate processing systems for the treatment of inorganic and aqueous waste. Chemicals present in these wastes will be (Monenco 1987a):

- Destroyed or stabilized within the physical/chemical plant;
- Separated for combustion in the rotary kiln incinerator;
- Separated and solidified for disposal in an engineered landfill; or
- Discharged in the liquid effluent sent to an evaporation unit.

In addition to the treated residues from the physical/chemical plant, the engineered landfill will also receive salt, ash, and slag from the incinerator, and some solid residues from off-site generators. The salt and ash, and, if necessary, the residues from off-site, will be stabilized in a solidification plant before being placed in the landfill.

In the risk assessment, the chemical composition of all wastes to be received by the proposed OWMC Facility was characterized using the CANVIRO data base (CANVIRO 1985), augmented by the Franklin data base for waste oil (Franklin Associates 1985). As discussed in the main report, only sparse data were available with respect to specific, well-defined organic contaminants in the inorganic residues sent to the landfill, and in the inorganic and aqueous organic wastes received by the physical/chemical treatment plant. There were, therefore, limited data with which to predict atmospheric emissions from the evaporator stack and contaminant concentrations in leachate generated in the engineered landfill.

Due to the limitations of the CANVIRO data base, the engineering consultants to OWMC have reviewed an alternative data base to provide additional information with which to estimate evaporator emissions and leachate concentrations (Monenco 1987b). This data base was compiled by the United States Environmental Protection Agency using the results of industrial wastewater analyses (USEPA 1983). The results of the work conducted by the engineering consultants is summarized in this appendix.

M.2 EVAPORATOR EMISSIONS

The results of the analysis of potential evaporator emissions, as conducted by the engineering consultants to OWMC (Monenco 1987b), are summarized in Table M-1. As shown in the table, the predicted maximum off-site concentrations in air for the chemicals examined are significantly lower than the human health-based "criterion" concentrations developed for the study. In interpreting the results of the analysis, it should be recognized that only inhalation exposure was considered in developing the "criterion" concentrations. Furthermore, the "criterion" concentration is applicable only to exposures to individual chemicals. The potential additive risks from exposures to contaminant mixtures have not been taken into account. The analysis does, however, indicate that evaporator emissions of the chemicals examined will not pose a significant risk to public health.

M.3 LANDFILL LEACHATE

The results of the analysis of potential chemical concentrations in leachate, as conducted by the engineering consultants to OWMC (Monenco 1987b), are summarized in

TABLE M-1

ESTIMATE OF EVAPORATOR EMISSIONS
(Basis: 150 000 t/a Delivered Waste)

	Average Contaminant Discharge Rate from Evaporator[1] (g/sec)	Maximum Off-Site Concentration in Air[2] (ug/m^3)	"Criterion" Concentration[3] (ug/m^3)	ADI/UCR Reference
Pesticides				
- Aldrin	1.78×10^{-9}	8.4×10^{-10}	2.7×10^{-4}	CAG 1985
- Lindane	1.78×10^{-10}	3.9×10^{-10}	0.91	USEPA 1986
PCBs				
- Aroclor 1242	4.95×10^{-10}	2.3×10^{-10}	7.0×10^{-4}	CAG 1985
Halogenated Aliphatics and Methanes				
- Methylene Chloride	4.75×10^{-4}	2.2×10^{-4}	0.22	CAG 1985
- Carbon Tetrachloride	3.86×10^{-6}	1.8×10^{-6}	2.3×10^{-2}	CAG 1985
Trichloroethylene	7.42×10^{-6}	3.5×10^{-6}	0.28	CAG 1985
- Chloroform	8.91×10^{-6}	4.2×10^{-6}	3.8 E-02	CAG 1985
Halogenated Ethers				
- Bis(2-chloroethyl) Ether	2.97×10^{-5}	1.4×10^{-5}	2.7 E-03	CAG 1985
Aromatics and Chlorinated Aromatics				
- Benzene	1.33×10^{-5}	6.3×10^{-6}	0.10	CAG 1985
- Hexachlorobenzene	1.24×10^{-7}	5.8×10^{-8}	1.8 E-03	CAG 1985
Polycyclic Aromatics				
- Acenaphthene	1.78×10^{-6}	8.4×10^{-7}	2.6×10^{-4}	CAG 1985
- Benzo(k) Fluoranthene	5.94×10^{-8}	2.8×10^{-8}	2.6×10^{-4}	CAG 1985
Phthalate Esters				
- Butyl Benzyl Phthalate	2.67×10^{-7}	1.3×10^{-7}	7.5	NTP 1982
Nitrosamines and Miscellaneous Organics				
- Acrylonitrile	7.43×10^{-5}	3.5×10^{-5}	1.3×10^{-2}	CAG 1985
Phenolics				
- 2,4,6-Trichlorophenol	5.94×10^{-7}	2.8×10^{-7}	0.15	CAG 1985
- 2-Chlorophenol	2.97×10^{-6}	6.6×10^{-6}	2.20	USEPA 1984 (Analogy to 4-chlorophenol)

TABLE M-1 (continued)

ESTIMATE OF EVAPORATOR EMISSIONS
(Basis: 150 000 t/a Delivered Waste)

	Average Contaminant Discharge Rate from Evaporator[1] (g/sec)	Maximum Off-Site Concentration in Air[2] ($\mu g/m^3$)	"Criterion" Concentration[3] ($\mu g/m^3$)	ADI/UCR Reference
Inorganics				
- Arsenic	2.64×10^{-9}	1.2×10^{-9}	2.0×10^{-4}	CAG 1985
- Beryllium	6.03×10^{-10}	2.8×10^{-10}	1.2×10^{-3}	CAG 1985
- Chromium	3.02×10^{-9}	1.4×10^{-9}	7.4×10^{-5}	CAG 1985
- Mercury	4.46×10^{-11}	9.9×10^{-11}	6.1	EPA 1986

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[1] Source: Monenco 1987b.

[2] Based on an annual-averaged dispersion factor of 0.47 (ug/m^3)/g/s) for carcinogens and a 2-week averaged dispersion factor of 2.21 (ug/m^3)/(g/s) for non-carcinogens (ORF 1987). The symbol "ug" stands for micrograms; 1.0 ug = 1×10^{-6} g.

[3] For non-carcinogens, "criterion" concentrations are based on Acceptable Daily Intake (ADI) values. For carcinogens, "criterion" concentrations have been calculated using Unit Cancer Risk (UCR) values and a one-in-one-million cancer risk level. In calculating the "criterion" concentration, it was assumed that the exposed individual is a 70 kg adult with a breathing rate of 23 m^3/d.

Table M-2. For several carcinogenic chemicals, the predicted contaminant concentration in leachate exceeds the human health-based "criterion" concentrations developed for the study. For example, the predicted leachate concentration for benzene is approximately ten time higher than the "criterion" concentration. For non-carcinogens, such as toluene and xylene, the leachate concentrations are generally near or below the "criterion" concentrations.

In order to estimate the potential for significant ground water contamination, it is necessary to evaluate the behavior of chemicals as they pass through the clay layer toward the bedrock aquifer. If contaminant concentrations are not reduced during transport of leachate to the aquifer, levels in the ground water could eventually approach those in the leachate. As discussed in Chapter 6, however, it is expected that various attenuation mechanisms, such as dilution, adsorption, dispersion, and degradation, will lead to signficant reductions in leachate concentrations over time.

It has been estimated by the hydrogeology consultants to OWMC that breakthrough of even those chemicals which do not adsorb strongly to clay will not occur for thousands of years (Gartner Lee 1987). For those chemicals which do adsorb strongly, e.g., PCDD/PCDF, at least hundreds of thousands of years will pass before the ground water could be significantly impacted.

TABLE M-2

ESTIMATE OF CONTAMINANT CONCENTRATIONS IN THE LANDFILL LEACHATE

(Basis: 150 000 t/a of Delivered Waste and
113 300 t/a of Solidified Mass to Landfill)

Contaminant	Contaminant Concentration in Leachate[1]	"Criterion" Concentration[2] (mg/l)	ADI/UCR Reference	Comments
Pesticides				
- 4,4-DDE	1.9 E-06	1.0 E-04	CAG 1985 (Analogy to DDT)	Not identified in OWMC waste characterization data (by CANVIRO) as going to physical/chemical or solidification.
PCBs				
- Aroclor 1242	2.0 E-06	8.1 E-06	CAG 1985	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.
Halogenated Aliphatic HC				
- 1-1-1 Tricholorethane	9.5 E-02	1.0	McNutt 1975	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.
- Trichloroethylene	1.4 E-02	3.2 E-03	CAG 1985	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.
- Methylene Chloride	4.8 E-03	2.5 E-03	CAG 1985	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.

TABLE M-2 (continued)

ESTIMATE OF CONTAMINANT CONCENTRATIONS IN THE LANDFILL LEACHATE

(Basis: 150 000 t/a of Delivered Waste and
113 300 t/a of Solidified Mass to Landfill)

Contaminant	Contaminant Concentration in Leachate[1]	"Criterion" Concentration[2] (mg/l)	ADI/UCR Reference	Comments
Halogenated Ether				
- Bis(2-chloroethyl) Ether	4.0 E-04	3.1 E-05	CAG 1985	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.
Monocyclic Aromatic				
- Benzene	1.0 E-02	1.2 E-03	CAG 1985	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.
- Toluene	5.6	10.0	USEPA 1986	OWMC waste characterization data indicates toluene to physical/chemical and solidification.
- Xylene	7.5 E-03	36.0	Marks et al. 1982	OWMC waste characterization data indicates xylene to physical/chemical and solidification.
Phthalate Ester				
- Butyl Benzyl Phthalate	6.9 E-04	8.6 E-02	NTP 1982	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.
PAH				
- Acenaphthene	4.4 E-04	3.0 E-06	CAG 1985 (Analogy to B(a)P)	Not identified in OWMC waste characterization data as going to physical/chemical or solidification.

TABLE M-2 (continued)

ESTIMATE OF CONTAMINANT CONCENTRATIONS IN THE LANDFILL LEACHATE

(Basis: 150 000 t/a of Delivered Waste and
113 300 t/a of Solidified Mass to Landfill)

Contaminant	Contaminant Concentration in Leachate[1]	"Criterion" Concentration[2] (mg/l)	ADI/UCR Reference	Comments
Inorganics				
- Arsenic	1.0 E-02	1.4 E-02	USEPA 1986	Quantities based on OWMC waste characterization data.
- Cadmium	1.0 E-02	2.6 E-02	ENVIRON 1985	Quantities based on OWMC waste characterization data.
- Chromium	1.0 E-02	1.8 E-01	USEPA 1986	Quantities based on OWMC waste characterization data.
- Nitrate	140	35	USEPA 1986	Quantities based on OWMC waste characterization data.

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[1] Source: Monenco 1987b.

[2] For non-carcinogens, "criterion" concentrations are based on Acceptable Daily Intake (ADI) values. For carcinogens, "criterion" concentrations have been calculated using Unit Cancer Risk (UCR) values and a one-in-one-million cancer risk level. In calculating the "criterion" concentration, it was assumed that the exposed individual is a 70 kg adult ingesting 2 liters of water per day.

During the extended period of time required for chemicals to travel through the clay, it is expected that biological and/or chemical degradation processes will occur, thereby reducing the concentrations of many contaminants, particularly organics. The impacts of these degradation processes have not been rigorously evaluated due to gaps in available data. It has been possible, however, to examine the effect of these processes on ground water concentrations by assuming degradation half-lives for organic chemicals in the leachate.

The approach used to estimate the effect of degradation processes on contaminant concentrations in the aquifer is described in detail in Chapter 6. Two cases were examined:

Case 1: Low molecular weight organics were assumed to have a half-life of 50 years; high molecular weight organics, a half-life of 5 000 years.

Case 2: Low molecular weight organics were assumed to have a half-life of 250 years; high molecular weight organics, a half-life of 25 000 years.

In both cases, the period of time necessary to reach the ground water was assumed to be 1 000 years for low molecular weight organics and 100 000 years for high molecular weight organics, values,that are consistent with estimates developed by the hydrogeology consultants (Gartner Lee 1987). A dilution factor of 3.0 relative to leachate concentrations was also assumed in both cases (Gartner Lee 1987).

The results of the study, summarized in Table M-3, indicate that even relatively long degradation of half-lives result in significant reductions in chemical concentrations in the aquifer. For almost all chemicals examined, ground water concentrations are below the "criterion concentrations" even if very long half-lives are assumed.

It should be recognized that there is considerable uncertainty in predicting the half-lives for most organic chemicals, and that the degradation byproducts may be relatively toxic in some cases. Research to more accurately quantify the impact of the various attentuation mechanisms is currently being performed by OWMC.

TABLE M-3

EFFECT OF CHEMICAL AND BIOLOGICAL DEGRADATION ON CHEMICAL CONCENTRATIONS IN THE AQUIFER[1]

	Leachate Concentration (mg/L)	Ground Water Concentration (mg/L) Case 1[2]	Case 2[3]
Low Molecular Weight Organics			
1,1,1 Trichloroethylene	9.5 E-02	3.0 E-08	2.0 E-03
Trichloroethylene	1.4 E-02	4.5 E-09	2.9 E-04
Methylene Chloride	4.8 E-02	1.5 E-09	1.0 E-04
Bis (2-chloroethyl) Ether	4.0 E-04	1.3 E-10	8.3 E-06
Benzene	1.0 E-02	3.2 E-09	2.1 E-04
Toluene	5.6	1.8 E-06	1.2 E-01
Xylene	7.5 E-03	2.4 E-09	1.6 E-04
High Molecular Weight Organics			
4,4 - DDE	1.9 E-06	6.1 E-13	4.0 E-08
Aroclor 1242 (PCBs)	2.0 E-06	6.4 E-13	4.2 E-08
Butyl Benzyl Phthalate	6.9 E-04	2.2 E-10	1.4 E-05
Acenaphthene	4.4 E-04	1.4 E-10	9.2 E-06

---

[1] All ground water concentrations assume a dilution of three-fold relative to leachate concentrations.

[2] Case 1: Low molecular weight organics: Travel time to the aquifer assumed to be 1 000 years. Degradation half-life assumed to to 50 years.
High molecular weight organics: Travel time to the aquifer assumed to be 100 000 years. Degradation half-life assumed to be 5 000 years.

[3] Case 2: Low molecular weight organics: Travel time to the aquifer assumed to be 1 000 years. Degradation half-life assumed to to 250 years.
High molecular weight organics: Travel time to the aquifer assumed to be 100 000 years. Degradation half-life assumed to be 25 000 years.

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APPENDIX N

HUMAN HEALTH RISKS ASSOCIATED
WITH THE OPERATION OF A
300 000 t/a OWMC FACILITY

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APPENDIX N

RISKS ASSOCIATED WITH THE OPERATION OF A 300 000 t/a OWMC FACILITY

N.1 INTRODUCTION

As part of the Phase 4B Risk Assessment, an analysis was undertaken to estimate the potential human health risks associated with emissions from an OWMC facility with a capacity of 300 000 t/a. At a facility capacity of 300 000 t/a, the following changes are expected to occur:

- The quantity of waste received at the facility will double;
- Two incinerator units will be available for the destruction of organic waste;
- Fugitive emissions from the waste storage tanks will be vented to the incinerator for destruction. At a capacity of 150 000 t/a, emissions to the atmosphere through a scrubbing unit would occur when the incinerator unit is not in operation. With two incinerator units, however, no fugitive emissions from the waste storage tanks will occur.
- Atmospheric dispersion of incinerator stack emissions will increase. Ambient air concentrations can be estimated using the following dispersion factor for two incinerator units (ORF 1987):

	Dispersion Factors (ug/m^3)/(g/s)	
	150 000 t/a	300 000 t/a
Average annual	0.064	0.04
Maximum 2-week	0.61	0.40
Maximum 1-hour	34.5	23.6
Maximum 2-minute	69.0	47.2

The effects of these changes in facility operations on the risks posed by the Proposed facility are discussed in this Appendix.

N.2 CHRONIC EXPOSURES

The potential human health risks posed by the following routine emissions were evaluated in the risk assessment:

- Incinerator stack emissions;
- Evaporator stack emissions;
- Leachate from the engineered landfill; and
- Fugitive emissions associated with
  - bulk waste unloading and transfer,
  - routine leaks and spills,
  - drum handling,
  - waste storage, and
  - physical/chemical treatment.

The methodologies used to estimate lifetime cancer risk estimates and chronic Adjusted Margins of Safety (AMOS) values corresponding to the routine operation of a 300 000 t/a facility were the same as presented in this report for a

150 000 t/a facility. The cancer risk estimates for a 300 000 t/a facility are presented in Tables N-1 and N-2. Table N-1 reports cancer risk estimates based on the assumption that polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/PCDF) are no-threshold carcinogens. Table N-2 presents the cancer risk estimates assuming that PCDD/PCDF are threshold carcinogens. Tables N-3 through N-5 present the adjusted AMOS values for chronic exposures to non-carcinogens from the three atmospheric emissions sources. The risks associated with the 300 000 t/a facility are less than twice those for a 150 000 t/a facility since greater dispersion of incinerator stack emissions would be expected (ORF 1987), and fugitive emissions from the waste storage tanks will be essentially eliminated.

The risks associated with the release of leachate from the engineered landfill have not been evaluated in the current assessment. Due to a lack of information on the environmental fate and transport of chemicals in the leachate, it was not possible to estimate the magnitude of human exposure that may occur. It was possible, however, to estimate chemical concentrations in the leachate (see Chapter 6 and Appendix C). The leachate concentrations associated with the operation of a 300 000 t/a facility would be the same as estimated for a 150 000 t/a facility, although twice as much leachate would be produced.

TABLE N-1

CANCER RISK ESTIMATES[1]
(300 000 t/a Facility)
Case 1: PCDD/PCDF assumed to be no-threshold carcinogens[2]

	Incinerator Emissions	Evaporator Emissions	Fugitive Emissions
Inhalation[3]	3×10^{-7}	2×10^{-8}	2×10^{-6}
Beef	1×10^{-6}	2×10^{-8}	9×10^{-9}
Milk	5×10^{-7}	3×10^{-8}	4×10^{-9}
Produce	9×10^{-8}	4×10^{-8}	4×10^{-7}
Pork	1×10^{-7}	7×10^{-9}	1×10^{-9}
Poultry	2×10^{-7}	1×10^{-8}	1×10^{-9}
Eggs	1×10^{-7}	8×10^{-9}	6×10^{-10}
Cistern	5×10^{-8}	2×10^{-9}	1×10^{-8}
Pond	3×10^{-8}	4×10^{-8}	1×10^{-7}
Soil Ingestion	1×10^{-9}	8×10^{-11}	6×10^{-9}
Soil Contact	6×10^{-9}	3×10^{-10}	2×10^{-9}

---

[1] Expressed as the probability of developing cancer. Thus, if the lifetime cancer risk is 1×10^{-6}, an exposed individual has a one-in-one-million chance of developing cancer; at a risk level of 1×10^{-7}, the individual has a one-in-ten-million chance of developing cancer.

[2] Polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) are assumed to be no-threshold carcinogens (see Appendix G). The toxicity of the PCDD/PCDF mixture has been estimated using the EPA toxic equivalency scheme (see Appendix H).

[3] Ten percent of the chromium assumed to be emitted in the hexavalent form (CrVI).

TABLE N-2

CANCER RISK ESTIMATES[1]
(300 000 t/a Facility)
Case 2: PCDD/PCDF assumed to be threshold carcinogens[2]

	Incinerator Emissions	Evaporator Emissions	Fugitive Emissions
Inhalation[3]	3×10^{-7}	2×10^{-8}	2×10^{-6}
Beef	2×10^{-7}	2×10^{-8}	9×10^{-9}
Milk	2×10^{-7}	3×10^{-8}	4×10^{-9}
Produce	7×10^{-8}	4×10^{-8}	4×10^{-7}
Pork	6×10^{-8}	7×10^{-9}	1×10^{-9}
Poultry	9×10^{-8}	1×10^{-8}	1×10^{-9}
Eggs	5×10^{-8}	8×10^{-9}	6×10^{-10}
Cistern	2×10^{-8}	2×10^{-9}	1×10^{-8}
Pond	2×10^{-7}	4×10^{-8}	1×10^{-7}
Soil Ingestion	7×10^{-10}	8×10^{-11}	6×10^{-10}
Soil Contact	3×10^{-9}	3×10^{-10}	2×10^{-9}

---

[1] Expressed as the probability of developing cancer. Thus, if the lifetime cancer risk is 1×10^{-6}, an exposed individual has a one-in-one-million chance of developing cancer; at a risk level of 1×10^{-7}, the individual has a one-in-ten-million chance of developing cancer.

[2] Polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) are assumed to be threshold carcinogens (see Appendix G). The toxicity of the PCDD/PCDF mixture has been estimated using the OMOE toxic equivalency scheme (see Appendix H).

[3] Ten percent of the chromium assumed to be emitted in the hexavalent form (CrVI).

TABLE N-3

AGGREGATED AMOS VALUES: EXPOSURE TO INCINERATOR EMISSIONS
(300 000 t/a Facility)

Exposure Pathway	Target Organ or System					
	Respiratory	Liver	Reproduction	Kidney	Blood	CNS*
Inhalation	1×10^5	5×10^2	6×10^2	1×10^4	3×10^4	6×10^2
Beef	2×10^7	2×10^3	2×10^2	2×10^2	2×10^2	6×10^4
Milk	4×10^9	1×10^3	3×10^2	2×10^4	3×10^2	3×10^4
Pork	2×10^8	1×10^4	7×10^2	1×10^3	1×10^3	7×10^4
Poultry	1×10^9	8×10^4	7×10^2	3×10^4	5×10^4	9×10^4
Eggs	5×10^6	1×10^3	1×10^2	4×10^6	2×10^6	1×10^3
Produce	2×10^5	3×10^3	5×10^2	2×10^3	2×10^2	7×10^3
Soil Ingestion	1×10^8	8×10^4	2×10^5	2×10^6	2×10^4	1×10^5
Soil Contact	4×10^7	2×10^4	2×10^4	6×10^5	6×10^3	3×10^4
Pond	3×10^6	1×10^4	7×10^3	8×10^3	7×10^2	5×10^3
Cistern	5×10^5	4×10^2	8×10^2	1×10^2	6×10^1	5×10^5

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*Central Nervous System

TABLE N-4

AGGREGATED AMOS VALUES: EXPOSURE TO EVAPORATOR EMISSIONS
(300 000 t/a Facility)

Exposure Pathway	Target Organ or System					
	Respiratory	Liver	Reproduction	Kidney	Blood	CNS*
Inhalation	8×10^4	9×10^3	8×10^4	6×10^4	2×10^3	8×10^4
Beef	1×10^{11}	1×10^3	1×10^3	3×10^3	5×10^2	2×10^{11}
Milk	1×10^{11}	9×10^2	2×10^3	3×10^5	5×10^2	1×10^{11}
Pork	2×10^{10}	8×10^3	1×10^4	2×10^4	3×10^3	2×10^{10}
Poultry	3×10^9	3×10^5	4×10^5	1×10^6	1×10^5	3×10^9
Eggs	6×10^9	7×10^5	9×10^5	2×10^6	3×10^5	6×10^9
Produce	9×10^6	6×10^5	9×10^6	2×10^6	9×10^3	9×10^6
Soil Ingestion	3×10^{10}	2×10^7	1×10^{10}	9×10^8	7×10^5	3×10^{10}
Soil Contact	1×10^{10}	5×10^6	3×10^9	3×10^8	2×10^5	1×10^{10}
Pond	7×10^6	1×10^5	7×10^6	2×10^6	2×10^5	7×10^6
Cistern	8×10^9	3×10^6	1×10^8	3×10^6	1×10^6	8×10^9

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*Central Nervous System

TABLE N-5

AGGREGATED AMOS VALUES: EXPOSURE TO FUGITIVE EMISSIONS (300 000 t/a Facility)

Exposure Pathway	Target Organ or System					
	Respiratory	Liver	Reproduction	Kidney	Blood	CNS*
Inhalation	9×10^{1}	2×10^{1}	9×10^{1}	8×10^{1}	9×10^{1}	2×10^{1}
Beef	9×10^{8}	2×10^{3}	4×10^{8}	5×10^{7}	1×10^{8}	2×10^{3}
Milk	2×10^{8}	1×10^{3}	9×10^{7}	1×10^{7}	3×10^{7}	1×10^{3}
Pork	2×10^{6}	3×10^{3}	2×10^{6}	1×10^{6}	2×10^{6}	3×10^{3}
Poultry	3×10^{6}	5×10^{3}	3×10^{6}	2×10^{6}	3×10^{6}	5×10^{3}
Eggs	5×10^{6}	9×10^{3}	5×10^{6}	3×10^{6}	5×10^{6}	9×10^{3}
Produce	1×10^{2}	7×10^{1}	1×10^{2}	1×10^{2}	1×10^{2}	9×10^{1}
Soil Ingestion	1×10^{6}	5×10^{3}	1×10^{6}	1×10^{6}	1×10^{6}	5×10^{3}
Soil Contact	4×10^{5}	2×10^{3}	4×10^{5}	3×10^{5}	4×10^{5}	2×10^{3}
Pond	6×10^{3}	1×10^{3}	6×10^{3}	5×10^{3}	6×10^{3}	1×10^{3}
Cistern	9×10^{4}	2×10^{1}	6×10^{4}	5×10^{1}	1×10^{5}	1×10^{5}

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*Central Nervous System

N.3 ACUTE EXPOSURES

The potential human health effects associated with the following upset and accident scenarios were evaluated in the risk assessment:

- A tank farm fire;
- Mixing of incompatible wastes;
- Incinerator scrubber failure;
- pH excursion during the treatment of cyanide-containing wastes; and
- pH excursion during sulfide precipitation of heavy metals
- A transportation spill resulting in the release of wastes
  - onto the roadway, or
  - into surface water.

For each of these events except the incinerator scrubber failure, the magnitude of human exposures would likely be the same for a 300 000 t/a facility as for a 150 000 t/a facility. This conclusion is based on the assumption that the expansion in capacity would be accomplished through an increase in the number of vehicles and pieces of equipment (e.g., storage tanks) at the site rather than an increase in the size of the vehicles and equipment. The frequency of these events would, however, be expected to double as shown in Table N-6.

For the incinerator scrubber failure, it is possible that potential magnitude of exposures would increase as a result of

TABLE N-6

FREQUENCY OF UPSET AND ACCIDENT EVENTS
(300 000 t/a Facility)

Event	Estimated Frequency
Tank Farm Fire	
• Segregated Tanks	Once in 350 to 650 years
• Unsegregated Tanks	Once in 50 years
Transportation Spill onto a Roadway	
• Any Spill	Once in 5 years
• Spill of potentially hazardous chemicals	Once in 65 years
• Spill of the most potentially hazardous chemicals	Once in 660 years
Transportation Spill into 20 Mile Creek	
• Average spill of any waste	Once in 440 years
• Large spill of any waste	Once in 4 400 years

the expansion to a 300 000 t/a facility. The increase in exposure would be related to the increase in emissions: the upset that would cause one scrubber to fail may cause the other to fail as well, thus doubling the estimated emission rate. This increase in emissions would be offset to a degree by the predicted increase in atmospheric dispersion for the expanded facility. For this event, the frequency would be the same for the 150 000 t/a and 300 000 t/a facilities.

The acute Adjusted Margins of Safety (AMOS) values corresponding to an incinerator scrubber failure at a 300 000 t/a facility are presented in Table N-7. As shown in the table, the Acute Safe Exposure Level (ASEL) values would be exceeded for hydrogen chloride and sulfur dioxide. The Short Term Public Limit (STPL) values would be exceeded only for hydrogen chloride. The STPL for hydrogen chloride is, however, based on a 10 minute exposure period, whereas the duration of exposure for the scrubber failure would be approximately 30 seconds. Since the magnitude of the irritating effects for hydrogen chloride is likely to be a function of both concentration and time, major adverse health effects would not be expected for a 30 second exposure. The only human health effects likely to occur as the result of an incineratory scrubber failure at the 300 000 t/a facility would therefore be slight odors or minor irritation experienced by sensitive members of the exposed population.

TABLE N-7

AMOS VALUES ASSOCIATED WITH AN INCINERATOR SCRUBBER FAILURE (300 000 t/a Facility)

Contaminant	AMOS[1] (Based on ASEL)	AMOS[1] (Based on STPL)
Hydrogen Chloride	0.33	0.73
Hydrogen Fluoride	7.3	--
Nitrogen Oxides	1.1	--
Sulfur Dioxides	0.042	1.2

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[1] Acute Adjusted Margin of Safety (AMOS) values based on toxicity data from Chapter 9, and chemical concentrations in air using the approach presented in Appendix D.

REFERENCES

Ontario Research Foundation (ORF). 1987. Phase 4B Site Assessment: Atmospheric Conditions (DRAFT). Prepared for Ontario Waste Management Corporation. June 1987.